

EMBOLISM AND THROMBOSIS
OF THE
MESENTERIC VESSELS

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EMBOLISM AND THROMBOSIS
OF THE
MESENTERIC VESSELS

BY

LESLIE B. C. TROTTER, M.A., B.C.(CANTAB.)

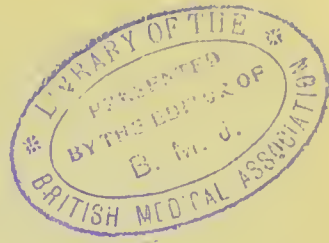
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PREFACE

THE subject of this work was suggested to the writer as being a suitable one for a thesis for the degree of M.D. Cambridge by the occurrence of two cases at University College Hospital during his tenure of office as house surgeon there in 1910. At the outset it seemed unlikely that sufficient material could be obtained from which generalisations might safely be made; but after further inquiry there proved to be a fairly large number of unreported cases in hospital post-mortem records alone, not to mention other cases which subsequently came under the author's own observation, and several series of cases already published on the Continent and in America.

The cases obtained from the London Hospital Pathological Institute have been accessible through the courtesy of Dr Turnbull, who very kindly placed the pathological reports at the disposal of the author. The present writer also wishes to express his thanks to Dr Andrewes for granting permission to examine museum specimens from two cases of venous thrombosis that were treated at St Bartholomew's Hospital, and to Dr Alexander Morison, Dr Batty Shaw, Dr J. B. Howell, Mr (now Sir) Rickman J. Godlee, Mr Raymond Johnson, Mr Wilfred Trotter, and Mr Morriston Davies for granting permission to publish their cases.

The author is also indebted to Dr Graham Smith for valuable advice and help in the publication of this thesis.

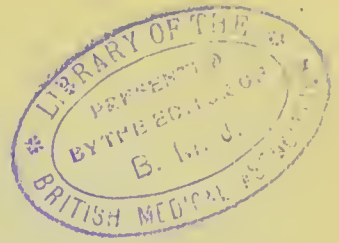
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LONDON, 1913.



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CONTENTS

	PAGE
INTRODUCTION	x
Historical	x
Material and arrangement of the paper	xi
SECTION I. ORIGINAL CASES	1
(1) Thrombosis following arterio-sclerosis in arcades of the superior mesenteric artery; anaemic infarction of part of the ileum	1
(2) Probable embolism of a branch of the superior mesenteric artery	6
(3) Perforated appendix; appendix abscess; 'primary' thrombosis of the superior mesenteric vein with most of its tributaries; haemorrhagic infarction of 15 inches of jejunum. Resection	8
(4) Atheromatous 'ulcer' in the aorta close to the origin of the superior mesenteric artery; 'ulcer' covered by thrombus extending into and completely occluding trunk of superior mesenteric. Haemorrhagic infarction of part of the intestine	16
(5) Acute appendicitis; removal of the appendix; 'primary' mesenteric venous thrombosis; local peritonitis; recovery	18
(6) Aneurism of the abdominal aorta, ruptured. Old, complete closure of superior mesenteric artery and coeliac axis, and partial closure of inferior mesenteric artery. Establishment of complete collateral circulation. No infarction or morbid changes in stomach or intestine	24
(7) Atheromatous 'ulcer' in the aorta, close to the origin of the superior mesenteric artery; 'ulcer' covered by thrombus; embolism of superior mesenteric artery. Haemorrhagic infarction of part of jejunum. Resection	32
SECTION II. CLASSIFICATION OF MESENTERIC VASOULAR OCCLUSIONS FROM THE STANDPOINT OF MORBID ANATOMY	34
Introduction to the Section	34
(a) Mesenteric Venous Occlusion	35
Portal Thrombosis	37
Secondary or Descending Mesenteric Venous Thrombosis	39
Primary Mesenteric Venous Thrombosis	39
Retrograde Venous Embolism	44
Results of Venous Thrombosis	45

	PAGE
(b) Arterial Thrombosis and Embolism	46
Introduction to Mesenteric Arterial Occlusion	46
(1) Thrombosis of the Mesenteric Arteries	50
Results of Mesenteric Arterial Thrombosis	54
Thrombosis of the Superior Mesenteric Artery alone	54
Thrombosis of the Superior and Inferior Mesenteric Arteries together	55
Thrombosis of the Inferior Mesenteric Artery alone	56
(2) Embolism of the Mesenteric Arteries	57
Source of the Embolus	57
Results of Embolism of the Mesenteric Arteries	59
Superior mesenteric alone	59
Inferior mesenteric alone	60
Embolism of both Mesenteric Arteries together	60
Purpura associated with Mesenteric Embolism	61
(c) Coexistent Occlusion of Arteries and Veins	62
Results	63
SECTION III. MORBID ANATOMY	64
(1) Vessels	64
(A) Veins	64
Distribution of the Thrombus	65
(B) Arteries	66
(C) Effects of Blood-Stasis upon Capillaries and Venules	68
(2) Mesentery and glands	68
(3) Intestines and their Contents	70
(A) Haemorrhagic Infarction	70
Extent of the Infarct	71
Condition of the Infarcted Coil	72
Contents of the Infarcted Coil	73
Condition of the Coils above and below the Infarct	73
Morbidity Histology	74
(B) Anaemic Gangrene	75
Hypotheses regarding aetiology	76
Brief table of cases of Anaemic Infarction	77
(4) Peritoneum	78
(5) Bacteriology	79
SECTION IV. EXPERIMENTS ON ANIMALS	80
(1) Experimental closure of Arteries	80
(2) Experimental closure of Veins	85
(3) Experimental closure of Arteries and Veins together	87
Experimental resections of extensive segments of Small Intestine	89
SECTION V. DESCRIPTIVE ANATOMY	91
(1) Superior Mesenteric Artery	91
(2) Inferior Mesenteric Artery	92
(3) Anastomoses between the Mesenteric and other Arteries	92

Contents

ix

	PAGE
(1) Superior Mesenteric Vein	93
(2) Inferior Mesenteric Vein	93
(3) Anastomoses between the Portal and Caval Systems	93
SECTION VI. SYMPTOMATOLOGY	95
Statistics of 'acute' and 'chronic' cases	95
Sex and age incidence	96
General account of the Course and Symptoms of the Disease	96
Individual Symptoms and Signs	98
Clinical Types	105
Antecedent Cardiac or Vascular Disease	106
'Intermittent mesenteric claudication' ('Dyspragia intermittens angiosclerotica intestinalis')	107
Cases associated with purpuric manifestations	109
Antecedent Hepatic Disease	110
Infective processes in the female pelvic organs	111
Infective processes in the region of the appendix	112
Post-operative Mesenteric Thrombosis	113
Type of the Disease in which clinical manifestations are absent	114
SECTION VII. DIAGNOSIS	115
Differential Diagnosis from acute intussusception	117
from volvulus	118
from stenosing carcinoma of the colon	119
from internal strangulation	119
from other conditions causing acute intestinal obstruction after appendicitis	119
from acute pancreatitis	120
from severe haemorrhage in hepatic cirrhosis	121
Differential diagnosis between superior and inferior mesenteric vascular occlusions	121
Diagnosis of 'intermittent mesenteric claudication'	122
SECTION VIII. TREATMENT	123
Operations that have been performed, and results	123
Choice of anaesthetic, and other considerations	126
Wilms' operation	128
Removal of Causal Infective Focus	129
Prognosis	129
SECTION IX. CONCLUSIONS	131
BIBLIOGRAPHY	134
LIST OF CASES	142
Platc I illustrating Case III	<i>to face</i> p. 13
" II " " VI	" p. 30

INTRODUCTION

HISTORICAL

OCCCLUSION of the mesenteric vessels was regarded till comparatively recent years as a very rare condition. The first case reported was that of Tiedeman in 1843. Virchow in 1847 and 1854 published three cases, and towards the end of the next decade cases were recorded by Moos, Hegar and Chiene.

Not long afterwards, Panum's experiments upon the subject of embolism were carried out, and Litten published his classical work upon the ligation of vessels in the mesentery.

Since 1875 the experimental work initiated by Litten and Faber has been repeated with numerous modifications, but by no means constant results, by Cohnheim, Cohn, Kader, Tansini, Rydygier, Bolognesi, Zesas, Marek, Bier, and many others. During the same period the number of clinical observations has steadily increased.

In 1904, Jackson, Porter and Quinby in America were able to collect 214 cases, and smaller series have been published from time to time by other authors (Deckart, Neutra, Zesas, Mauclaire and Jacoulet, Roussel, etc.). There has been a good deal of unavoidable overlapping in the different series, the outcome possibly of special attention being devoted to different aspects of the disease.

With the increased study of mesenteric embolism and thrombosis further attention has been paid to allied conditions. Thus in Germany, Breuer, Schnitzler, and Orthner, and in France, Teissier, Sauv , and Lagane, have devoted particular attention to the clinical and pathological aspects of atheroma and arterio-sclerosis in the mesenteric arterial system, and the influence of these as factors in thrombosis.

The significance of endocarditis of rheumatic origin as the main cause of mesenteric embolism was emphasised by Gallavardin, while Talke drew attention to the aetiological relationship between mesenteric embolism and certain forms of purpura.

Two series of cases were collected by Borrmann and Lissauer respectively, who established the relationship between alcoholic cirrhosis of the liver and syphilitic perihepatitis on the one hand, and thrombosis in the portal system on the other.

Mesenteric venous thrombosis and thrombo-phlebitis were considered to be particularly liable to follow appendicitis by Polya in Germany and Gerster in America: their conclusions have been amply borne out by the cases of that nature collected in the present series.

In this country no attempt has been made to deal with the subject in a comprehensive and at the same time a detailed review. Investigations have been undertaken, however, in America and on the Continent: some of these, admirable as they are, do not deal with the material up to a sufficiently recent date to be regarded as giving the present state of our knowledge, others approach the subject from some special point of view, and many being based upon a study of only some of the available cases embody conclusions which a fuller knowledge of the facts has shewn to be inexact.

It therefore appeared to the present writer that a review of the matter might be of value provided it was comprehensive of all aspects of the subject and of all the available material. An effort has thus been made in this paper to deal with the subject in as complete a manner as possible, though this has been rendered increasingly difficult by the wealth of current literature: scarcely a month passes without one or more contributions to the subject appearing in Continental or American periodicals.

ARRANGEMENT OF THE PAPER

An attempt has been made to consult as many as possible of the original works and series of cases. It has not been thought necessary to publish all the material thus obtained, but an analysis of the cases has been compiled and deposited in the Library of the Medical Schools at Cambridge.

The statistics given in this paper have been compiled from the analysed records at Cambridge : owing to the scantiness of the data concerning many of the cases, the statistics are not so complete as might be desired. In a few instances the statistics given by other writers have been quoted.

The results of experimental work have been obtained exclusively from the literature, no original experiments having been undertaken. Unfortunately much of the research work has been published in Italian and Russian, and on this account the reviews of other authors have been utilised.

In order not to overload the text the names of authors have been excluded as much as possible, and indicated in footnotes instead. An exception to this rule has been made in dealing with operative treatment : in this case the names have been retained in the text to prevent confusion.

At the beginning of the paper a full account is given of seven hitherto unpublished cases of mesenteric vascular obstruction. A few drawings have been made to illustrate cases Nos. III and VI.

The second section of the paper has been devoted to a classification of the disease from the standpoint of its morbid anatomy. A detailed account of the morbid anatomy and histology is given in the next section.

Section No. IV comprises a review of the more important experimental work that has been done in connection with circulatory disturbances in the mesentery, together with a brief account of the results of experimental resection of large portions of small intestine.

For the sake of clearness in the discussion of the subject it has been considered necessary to give a short account in section No. V of the descriptive anatomy of the mesenteric vessels and their anastomoses with other systems.

An attempt has been made in the next section to make a clinical classification of the lesions which result from mesenteric circulatory disturbances brought about by embolism or thrombosis.

The questions of diagnosis and treatment occupy the next two sections, and the last consists of a brief summary of conclusions previously considered.

SECTION I

ORIGINAL CASES

CASE I

Thrombosis following arterio-sclerosis in arcades of the superior mesenteric artery; anaemic infarction of part of the ileum. Under the care of Mr (now Sir) RICKMAN J. GODLEE.

HISTORY. The patient was admitted as an emergency case to University College Hospital at 12.30 a.m., Feb. 10th, 1910, complaining of some constipation and vomiting for 10 days, and severe abdominal pain, and distension for 3 or 4 days. The vomited material had been yellow in colour, and watery, and contained no blood. No blood had been seen in the stools.

PREVIOUS HISTORY. The patient had been subject to attacks of diarrhoea for 24 years. On one occasion about 15 years ago she had had to call in a doctor at night on account of a severe attack.

During recent years the diarrhoea had been better. There had been no other gastro-intestinal troubles.

PRESENT STATE (Feb. 10th, on admission). The patient is a thin, poorly nourished woman of 48. She is in considerable distress, is breathing with some difficulty, and vomiting some watery, inoffensive, yellowish material. There is severe, generalised, abdominal pain.

The temperature is 100·2, and pt. is sweating profusely. The pulse is 136 per minute. There is a short, hacking cough, associated with scanty purulent expectoration. There is marked clubbing of the fingers.

Chest: There is deficiency of resonance over both bases, and coarse râles are heard all over both sides, back and front.

No murmurs are heard over the heart.

Alimentary system: The tongue is coated with yellowish

curd-like fur. The abdomen is very distended, and the area of liver dulness is slightly raised. There is dulness over the whole abdomen, and generalised, extreme tenderness.

The vomited matter is free from blood.

No diagnosis was made, and grave doubts being entertained as to whether the patient could stand a general anaesthetic owing to the condition of the chest, it was decided to explore the abdomen under local anaesthesia.

OPERATION. Mr Raymond Johnson (Feb. 10th, 2 a.m.).

A strip of the abdominal wall was rendered anaesthetic with eucaine, and a vertical incision made below the umbilicus. When the peritoneal cavity was opened a few oz. of clear, straw-coloured fluid, but no gas, escaped. The small intestine did not appear to be abnormally distended, but there were a few recent adhesions between adjacent coils. There was no abnormal discolouration of the intestines, nor was there any evidence of tubercular peritonitis.

After a few oz. of the fluid had been allowed to escape, and all the intestine within reach of the finger had been examined, the abdomen was closed. More could not be attempted on account of the extremely collapsed condition of the patient.

After the operation, the temperature was 98·4, the pulse 128 per min., feeble, and the respirations 40.

PROGRESS. The patient vomited once after the operation. She was allowed water only to drink.

Feb. 10th. Pt. complains of abdominal pain. No urine has been passed since the operation. The respirations are quieter and not so hurried. The signs in the chest remain unchanged. Pt. is being kept propped up in bed, and given albumin water and peptonised milk by the mouth in feeds of $\frac{1}{2}$ oz. every hour. The abdomen continues to be very tense, and no result followed a soap enema ($1\frac{1}{2}$ pint).

Feb. 11th. Pt. did not sleep last night in spite of the administration of paraldehyde dr. 1. Urine was passed normally.

The abdomen is still tense, tender, and painful. Pt. vomited twice and retched constantly during the morning. No result followed a turpentine enema.

At 11 a.m. she was given calomel gr. 1, and a rectal tube was

introduced to relieve distension. No flatus or faecal matter was passed till the afternoon.

During the day pt. received a subcutaneous normal saline infusion (4 pints), and small (2 oz.) feeds of peptonised milk and barley water every 2 hours. She did not vomit during the day.

At 10 p.m., T. 98, P. 124, and R. 28.

Feb. 12th. The abdomen is much less tense to-day, and pt. is much more comfortable. The râles on the right side of the chest have cleared up, but there is still dulness at the left base where some friction sounds are heard.

During the night pt. slept $2\frac{1}{2}$ hours only, after potassium bromide gr. 20, and chloral hydrate gr. 10.

At 12.30 a.m. she received another infusion of saline solution (3 pints) subcutaneously in the thighs, and at 2, 4, 6 and 8 a.m. she was given magnesium sulphate dr. 1 (by the mouth). During the day the bowels were open four times, the stools being loose and digested, and containing no blood. Urine was passed.

There has been no vomiting to-day.

Diet: Milk and barley water (2 oz. of each) every 2 hours.

At 10 p.m., T. 98, P. 124, and R. 24.

Feb. 13th. General condition is better. The abdomen is much less tender and painful. Pt. only dozed last night after potassium bromide and chloral, and did not sleep properly. She slept a little during the day.

There has been no more vomiting.

At 4 p.m. T. rose to 102·4, but pt. complained of no pain.

Diet: Milk and barley water ($2\frac{1}{2}$ oz. of each) every 2 hours.

At 10 p.m., T. 101·4, P. 136, and R. 36.

Feb. 14th. Last night pt. became delirious. The temperature rapidly dropped from 101·4 to 98, and at 5 a.m. to 95, when pt. became very restless. The respirations became very hurried and rattling, and the pulse-rate rose to 140.

At 5.45 a.m. she was semi-comatose. She was given a subcutaneous saline infusion ($1\frac{1}{2}$ pints) in the right arm and breasts, and $\frac{1}{2}$ oz. of brandy.

Pt. died at 7.30 a.m.

AUTOPSY. The body is that of a poorly nourished woman: laparotomy has been performed and the wound is healthy.

There is a large excess of clear fluid in the peritoneal cavity; the intestines are distended, and there is hyperaemia along the line of their mesenteric attachment. In the small intestine downwards from a point 18 inches above the ileo-caecal valve there is a segment about 9 inches long in which there are several areas of complete necrosis of the gut-wall; these patches vary from $\frac{1}{2}$ to $\frac{3}{4}$ inch in length, they are yellowish grey in colour, and one is on the point of yielding; they are adherent to neighbouring coils of intestine by recent lymph. The thickness of the walls of the affected coil shews but little change from the normal. There is no blood in the lumen of the intestine. The mesenteric arterial arcades corresponding with the affected coil are thrombosed.

The rest of the intestine is normal.

The liver is of normal size and consistence, and contains no suppurative focus. The portal vein, gall-bladder and bile-passages are normal.

The spleen is large and shews several areas of recent infarction.

The kidneys are of normal size and colour; the capsules are slightly adherent, and there are a few cysts; the cortex is reddened and tough. The renal vessels are normal.

The suprarenals are normal.

Chest: In each pleural cavity there is about a pint of clear fluid, containing large flakes of lymph. The air-passages are intensely hyperaemic, especially the smaller ones, and contain dirty, watery material.

The lungs: The upper lobes are emphysematous, and the lower very oedematous; there is some collapse but there are no patches of consolidation.

The intra-thoracic glands are normal.

The heart is normal in size, colour and consistence; the valves are all normal; there is no trace of endocarditis.

CONCLUSIONS.

This case is of particular interest both from the point of view of the clinical manifestations and of the morbid anatomy.

History and Course: Previously to the terminal illness there had been a long history of diarrhoea unassociated with any other gastro-intestinal disturbances (so far as could be ascertained from

the patient or her husband). After the onset of the present illness the patient had complained of constipation, and not diarrhoea. After admission to hospital constipation was absolute for about 24 hours only. There was never any blood in the stools or in the vomited matter, though vomiting was frequent. The pain though severe was never agonising.

The course of the terminal illness extended over a period of 14 days, but in other respects the symptoms and physical signs somewhat resembled those met with in an acute case of anaemic gangrene described by Sprengel. In his case¹ there had been 'abdominal inflammation' for 20 years.

Morbid anatomy. In this case we have an example of anaemic infarction of a small portion of the lower ileum caused by thrombosis in the corresponding arterial arcades, without any involvement of the veins. The thrombosis was probably caused by chronic sclerosis of the affected vessels, the walls of which shew marked local thickening. The larger branches of the mesenteric artery shew no gross changes, but appear to be normal; the same statement applies to the smaller twigs radiating from the arcades. The thrombosis and sclerosis appear to be confined therefore to the arcades.

It is possible that the arterio-sclerosis was responsible for the very chronic history of diarrhoea given by the patient.

As regards the infarcted coil, there is no remarkable change in the thickness of the walls; they are however absolutely devoid of blood, and there is no blood in the lumen of the intestine.

The aetiology of this and other cases of anaemic infarction will be considered later (*vide* 'Morbid Anatomy').

¹ SPRENGEL. M. 38. Sudden acute abdominal pains while at work, Dec. 21st. Vomiting. Unchanged till 25th, when he improved: then worse again. No motion since 21st. 'Abdominal inflammation' over 20 years before, lasting 5 weeks.

Dec. 25th. P. 80—84, regular. Heart normal. Abdomen distended: dulness in L. side of abdomen: loud gurgling.

Op. Dec. 25th. Moderate amount of turbid red-brown fluid in abdomen: 10 cms. of ileum, 30—40 cms. from ileo-caecal valve extremely gangrenous, and tore when moved, discharging contents: sharply demarcated above and below, and sharp line in mesentery parallel to intestine. No ligature required during resection: ends brought out to surface.

Feb. 5th. Artificial anus closed by second operation.

Complete recovery. Note. Anaemic gangrene in this case attributed to simultaneous occlusion of art. and vein.

CASE II

Probable embolism of a branch of the superior mesenteric artery.
Under the care of Mr MORRISTON DAVIES.

M. 64. Admitted July 30th, 1908, for a septic condition of the hand.

July 31st. OPERATION. Extensive incisions made in palm of left hand.

Secondary haemorrhage developed and the patient was admitted to the ward. Haemorrhage stopped at once after admission.

Wounds all continued to drain freely. No collection of pus.

Aug. 10th. Wounds improving steadily though slowly. No accumulation of pus and no fresh haemorrhage. Irrigated daily with sterile water.

Aug. 15th. No tracking of pus, and wounds all draining freely; healing more rapidly. In spite of this thrombosis suddenly developed in all the cutaneous veins of the anterior part of the forearm. Hot fomentations applied.

Sept. 2nd. Thrombosis of veins of forearm subsiding. Veins have not been sufficiently infected apparently to break down, and there has been very little pain associated with the infective process.

Condition of palm is now improving daily.

Sept. 16th. Forearm and arm much improved. Wound on ulnar side of palm allowed to close. Sinus under anterior annular ligament still plugged lightly. Wound in centre of palm granulating.

Sept. 21st. Pt. this morning complained of *pain* in the *chest*, and later on had a *rigor*. Condition of forearm and hand is improving and is evidently not the cause of the rigor. Over the whole of the *base* of the *right lung* there is complete dulness on percussion and absence of all breath and adventitious sounds. Apex beat of heart not displaced.

Pt. has slight cough and this causes him pain over the right base: slight *haemoptysis*.

Exploratory needle inserted into right base drew off blood only.

Sept. 22nd. Pt. has had no further rigor. He feels better and the signs in the chest are already subsiding.

Sept. 23rd. Further improvement. Signs in chest disappearing rapidly.

Sept. 26th. Hand almost well, save for small sinus under annular ligament. No signs in chest.

Sept. 28th. Early this morning pt. developed *diarrhoea* and *tenesmus*, associated with great *pain* in the lower part of the *abdomen*. He then had a rigor followed by nausea and *vomiting*. No blood in vomit. Pt. looks very weak and exhausted. Tongue very dry and furred. *Diarrhoea* and *tenesmus* continue and pt. has lost control over his motions. Abdomen moves well but is somewhat distended and there is generalised tenderness over the whole of the lower part.

Sept. 29th. Vomiting is less: *diarrhoea* persists with loss of control. Tenderness not quite so marked. No signs of obstruction: abdomen moves well. Slight improvement in general condition of pt.

Sept. 30th. Improvement more marked. Vomiting has ceased but there is still *diarrhoea* with pain. No further signs have developed.

Oct. 3rd. *Diarrhoea* has ceased and pt. is now improving steadily.

Oct. 6th. Pt.'s bowels have acted more normally. Control over sphincter ani has returned.

Oct. 11th. Abdominal condition quite normal again. Hand almost completely healed. Pt. allowed to go home.

SUPPLEMENTARY NOTE. Nearly a year later pt. was treated in the Out-patient Dept. for an attack of *haematuria* which subsided within a day or two.

CONCLUSIONS.

Though some doubt exists regarding the correctness of the diagnosis, the march of events in this case was strongly suggestive of mesenteric embolism resulting from a small infarct of the lung.

The probable course of the disease was as follows: As a result of infection of the superficial veins of the forearm from a condition of sepsis in the hand, emboli were carried to the base of the right

lung. The infarct in the lung can have been only a small one, as the symptoms and signs completely subsided in 5 or 6 days. The infarct though small possibly contained septic organisms which led to its breaking down. The sudden acute pain in the lower abdomen and the diarrhoea, tenesmus and vomiting occurring as they did soon after signs of pulmonary infarction suggest that a small branch of the superior mesenteric artery had become blocked by an embolus from the lung. The view that the abdominal symptoms were embolic in origin derives a certain amount of support from the occurrence about a year later of signs suggestive of renal infarction.

The view given above seems to the present writer to be the most probable one; a less plausible explanation of the course of the disease would be that emboli from the veins in the arm reached the mesenteric artery through a patent foramen ovale; but in the face of definite signs in the lungs followed soon after by abdominal symptoms, combined with the absence of evidence as to the presence of a patent foramen ovale, we are scarcely justified in accepting the latter view.

CASE III

Perforated appendix; appendix abscess; 'primary' thrombosis of the superior mesenteric vein with most of its tributaries; haemorrhagic infarction of 15 inches of jejunum. Under the care of Mr MORRISTON DAVIES.

HISTORY. The patient was admitted as an emergency case to University College Hospital at 8 p.m., April 25th, 1910, complaining of severe abdominal pains, particularly in the left iliac region, and vomiting of 'coffee-ground' material.

17 days previously (i.e. on Friday, April 8th) he was seized with acute pain in the right iliac region; he did not vomit, and had no rigor. Some distension of the abdomen was present from the onset.

On Sat., April 9th, a doctor was called in. A milk diet and rest in bed were advised. The temperature was then 103.

During the following week (April 9th to 16th) the symptoms gradually subsided, and the patient got up on Sat. and Sun., April 16th and 17th.

On Mon., April 18th, he began to have some pain in the epigastric region, passing through between the shoulder-blades, of a boring character.

Since Mon., April 18th, he had been constipated, but the bowels had acted with aperients.

On Sun., April 24th, he vomited some clear watery material; and next day, before admission, he vomited 'coffee-ground' material several times.

The pain since the 18th had extended to the left iliac fossa. There had been no pain on micturition. The bowels had usually been regular. He had had no cough.

PREVIOUS HISTORY. He had complained of 'heart-burn' for 2 years. 15 years before he had had small-pox. The general health had been good.

PRESENT STATE (on admission). Pt. is a heavily built man of 60, and is obviously very ill. The face is ashy grey and covered with sweat, the expression anxious, and the features pinched. The tongue is dry and furred, and vomiting ('coffee-ground' material) is constant. The temperature is 99·2, the respirations are 38 per min., and the pulse 120 per min. and thready in character. The extremities are cold.

The abdomen is rather distended, tympanitic generally, but dull in the left flank. Pain and tenderness are present particularly in the left iliac region, and to a less extent in the lower part of the abdomen. There is no special tenderness in the right iliac region. No definite mass can be felt, though increased resistance is present in the left iliac region between the anterior superior spine of the ileum and the umbilicus. Nothing abnormal is felt per rectum. The liver dulness is present.

The heart and lungs are normal.

OPERATION. By Mr Morriston Davies. 9.30 p.m. Chloroform, followed by ether given by the 'open' method.

Under the anaesthetic a definite smooth, soft, cylindrical tumour could be felt in the lower part of the left loin, extending down into the left iliac fossa.

A vertical incision was made through the upper part of the right rectus. The peritoneal cavity being opened, oedematous omentum about $\frac{3}{4}$ inch thick was seen, and when this had been

displaced there oozed up some reddish brown fluid. The hand was introduced, and the mesentery was felt to be greatly thickened. There were some slight adhesions about the mesentery, and when these were broken down a considerable quantity of fluid of the same nature as that described above escaped. A soft doughy mass was felt in the left loin.

This mass proved to be a coil of dark purple coloured small intestine. The affected segment appeared to commence in the left loin and extend downwards along the iliac fossa to the bottom of the pelvis. It was drawn out with difficulty owing to the great thickness and rigidity of the mesentery. Above, the affected portion of the intestine merged gradually into normally coloured, very slightly distended but oedematous intestine; below, the transition was much more abrupt, and there was much less oedema, but the intestine had not the collapsed and shrunken appearance of that below a complete obstruction. The affected loop and its mesentery were in no way kinked or twisted, and there was no evidence of pressure on, or compression of the mesenteric vessels. The condition was obviously one of thrombosis.

The affected portion of bowel was beginning to lose its gloss, but there were no adhesions; it was soft and patulous.

The corresponding mesentery was ligatured and divided. Starting at a point $3\frac{1}{2}$ ft. from the duodeno-jejunal flexure, 27 ins. of jejunum was resected, including 15 ins. of deeply congested bowel, and a margin above this of 7 ins., and below of 5 ins. of healthy intestine. The mesentery where divided was very oedematous. The intestine at the upper point of division appeared to be perfectly healthy and in no way paralysed. An end-to-end anastomosis was made, the two ends being sutured with thread. The arteries in the mesentery supplying the affected intestine spurted freely when divided, but the veins were thrombosed and contained tarry-looking blood clot.

The abdominal wound was then closed.

PROGRESS. April 26th, 1 a.m. Pt. is fairly comfortable, but complains of a little soreness of the abdomen. He has vomited three or four times since the operation. The pulse is of fair volume, and the abdomen is not distended.

Pt. is being kept propped up in Fowler's position.

9 a.m. Pt. is more comfortable. The vomiting has stopped since 4 a.m. Neither flatus nor faeces have been passed. He is having sodii sulphat. dr. 1 hourly, up to 6 doses.

4 p.m. Pt. is feeling very uncomfortable. He has been vomiting 'coffee-ground' material since 1 p.m., and cannot retain his food or medicine. He complains that the bandage is too tight, looks very ill, and has a pinched, drawn expression. The vomit is ejected with very little force, and consists of 'coffee-ground' material. The abdomen is distended but the bandages are not tight. The respiration is chiefly thoracic. The pulse is 136 per min. and of small volume. Pt. is sweating profusely. Sodii sulphat. stopped.

5 p.m. Normal saline subcutaneous infusion (4 pints) given. Iced champagne ordered.

10 p.m. Pt.'s condition improved a little after the infusion, but is now worse again. He complains of severe pain in the abdomen, and is vomiting constantly. He cannot answer questions coherently, and groans incessantly. The abdomen is distended and immobile; there is no tenderness except over the operation wound. The pulse is uncountable, and very small in volume. The tongue is dry and black. Constipation is absolute.

11.30 p.m. Pt. was given a turpentine enema (containing $1\frac{1}{2}$ oz. of oil of turpentine in one pint of water) without result. No flatus was passed.

April 27th. Pt. is worse; he is quite collapsed, and the abdomen is still distended. Another enema was given at 9 a.m. with no result. Vomiting is incessant.

11 a.m. The stomach was washed out, and a great deal of 'coffee-ground' material removed. Pt. struggled a little at first. The general condition is very bad. The pulse is uncountable, and hardly to be felt at the wrists. The tongue is dry and black. Another subcutaneous infusion (4 pints) was given.

Pt. died at 1.45 p.m.

AUTOPSY (partial). The body is that of a stout, heavily-built man of 60. There is a considerable quantity of turbid blood-stained serum in the peritoneal cavity. The enteroanastomosis $3\frac{1}{2}$ ft. from the duodeno-jejunal flexure is quite healthy, and there are no adhesions in this region. The small intestine above the

anastomosis is rather distended, whereas that below is rather contracted; but both afferent and efferent coils appear to be quite healthy.

In the appendix region there is an abscess-cavity, completely shut off from the general peritoneal cavity by the mesentery of the terminal portion of the ileum and by adhesions. In the abscess there are about $\frac{3}{4}$ oz. of dirty yellow pus, and the appendix which firmly adherent to the parietal peritoneum by recent lymph.

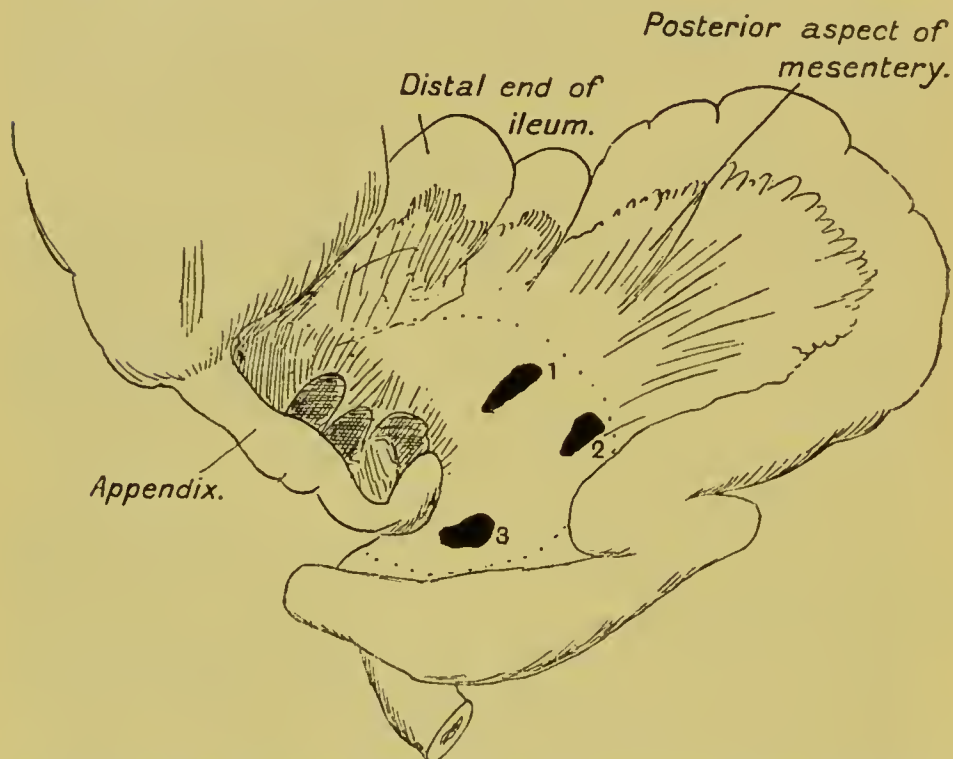


Fig. 1. Dotted line indicates junction of anterior and posterior walls of abscess cavity before reflection of coil of ileum and its mesentery. Area within dotted line indicates part of wall of abscess-cavity. 1, 2 and 3 indicate patches of engorgement in wall of abscess.

The appendix is very much thickened, and is directed towards the brim of the pelvis; its distal quarter is kinked, and at the junction of the 1st and 2nd thirds there is a large perforation.

On the upper and inner wall (that formed by the mesentery) of the abscess-cavity there are three small patches of reddish brown discolouration¹. (One of these patches will be referred to later.)

¹ See illustration (Fig. 1).

- A = portion of liver in region of termination of portal trunk.
 B = pancreas.
 C = duodenum.
 D = jejunum.
 E = ileum (divided to shew abscess-cavity).
 G = caecum.
 H = appendix.
 J = abscess-cavity.
 K = portal vein.
 L = point of entry of splenic vein (vein removed with anterior wall of portal vein) into portal.
 M = superior mesenteric vein filled with thrombus.
 O = permeable tributaries of superior mesenteric vein.
 P = right colic vein entering superior mesenteric.
 R = thrombosed tributaries with thickened walls corresponding with resected portion of jejunum.
 S = cut ends of jejunum above and below resected segment.



The caecum, the whole of the colon and the rectum are normal.

The whole of the mesentery and meso-colon are heavily loaded with fat; the former is greatly thickened and very oedematous, measuring in section $\frac{2}{3}$ to $\frac{3}{4}$ inch in places.

The lymphatic glands in the mesentery are slightly enlarged, but contain no haemorrhages.

The superior and inferior mesenteric arteries and their branches are normal, and contain no emboli or thrombi.

Practically the whole of the superior mesenteric vein and its main tributaries are thrombosed (see Plate I). The right colic vein however is quite free up to its outlet into the superior mesenteric trunk. The thrombus in the trunk extends further

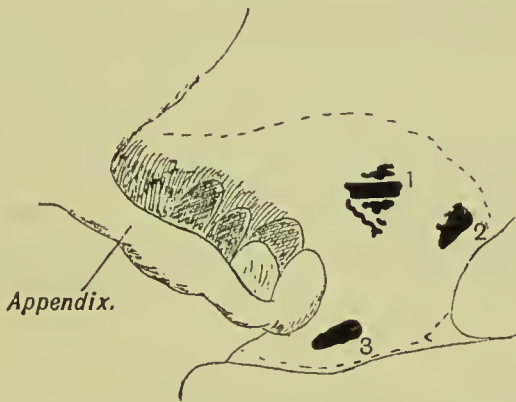


Fig. 2. 1. Thrombosed vein immediately beneath patch of engorgement.
2 and 3 as in Fig. 1.

than this point, but it is here quite loose and appears to be capable of allowing the passage of a certain amount of blood. At the periphery of the superior mesenteric venous system there is a fringe of permeable anastomosing arcades, except in the segment of mesentery corresponding with the infarcted portion of jejunum. Here, instead of the thrombus terminating about 1 or 2 ins. from the intestinal attachment, it has advanced right down into the finest radicles. Moreover, in this area the walls of the veins are very much thickened, and are extremely adherent to the surrounding fat and connective tissue; they are also rather tortuous, but this is probably due to retraction of the mesentery after resection of the infarcted segment of intestine.

The appendicular and other small veins in the region of the caecum are free from clot, but the origin of the superior mesenteric itself is thrombosed. This part of the vein¹ lies immediately over one of the three reddish brown patches in the abscess-wall mentioned above, and a minute thrombosed venule leads from the patch into the superior mesenteric at this point.

It seems clear therefore that this was the point of origin of the thrombosis.

The clot throughout the affected area is fairly uniform in structure; it is not stratified and shews no signs of breaking down, or of pus formation; it is slightly adherent to the intima at the junction of the 2nd large jejunal vein and the trunk, and also in the veins draining the infarcted area.

It would seem therefore that the upper jejunal region was the first to become affected after the actual onset of the process in the abscess-wall. The thrombosis probably spread downwards from the jejunal veins, and at the same time from the appendicular region, till finally the peripheral anastomosing channels and the right colic were the only veins left patent.

The intestine was able to maintain its vitality so long as these channels remained open, and it was only in the area where these also had become occluded that infarction took place.

The portal, splenic and inferior mesenteric veins are normal.

The liver shews no cirrhosis, and appears to be normal.

The spleen is not enlarged; the structure is normal.

The kidneys shew some fibrosis.

The pancreas is normal.

HISTOLOGICAL EXAMINATION.

Portions from six different parts of the thrombosed mesenteric vein and its tributaries were removed and examined histologically. A piece removed from the main tributary corresponding with the infarcted segment of intestine shewed marked inflammatory thickening of the adventitia; but apart from this, no pathological changes were found in any of the specimens examined, though they were all filled with recent blood-clot.

¹ See Fig. 2.

Portion of jejunum removed at operation.

The specimen measures about 27 ins. in length, and may be described under three headings, viz. the afferent coil, the infarcted middle portion, and the efferent coil.

(1) In the afferent segment the transition from normal healthy intestine to complete infarction is gradual. The diameter of the gut is increased, but is not so great as that of the infarcted portion below. The walls shew thickening from oedema, and engorgement with blood, but except at the lower end actual extravasations are absent. The mucosa is thickened but not necrotic.

(2) The middle portion shews definite haemorrhagic infarction. The coil is much thickened and of a purple-black colour. The thickening of the walls is due to extreme oedema, engorgement, and extravasation of blood. The mucosa is thickened, black in colour, and necrotic, but there is no ulceration. There is a quantity of black blood in the lumen. The thrombosed veins can be traced into the wall of this portion of the specimen, but not into that of the upper part of the afferent coil or of the efferent coil.

(3) The efferent coil is marked off sharply from the part above, and except for the diminution in diameter mentioned above, it appears to be fairly normal.

CONCLUSIONS.

As this case is fairly typical of a particular clinical group which we shall have to consider at a later stage (*vide* 'Symptomatology'), only the outstanding features will be dwelt upon here.

The course of the disease from the onset of appendicitis till death from infarction and toxæmia may be divided into four separate periods:—(1) A period of 5 or 6 days during which the patient was suffering from symptoms of acute appendicitis. The symptoms gradually subsided, and there followed (2) A period of 2 or 3 days during which there were practically no symptoms. (3) About the 10th day after the onset there was a recrudescence of symptoms, due this time probably to the development of mesenteric venous thrombosis. During the next 7 days the manifestations gradually increased in severity till, (4) 17 days after the onset, when they became extremely grave; by this time

(i.e. a few hours before admission to hospital) infarction of the intestine had developed as a result of invasion of the venous radicles by blood-clot.

If we may assume that the thrombosis began directly after the latent period referred to above, the process took an unusually long time to spread into the radicles in the jejunum. By comparison with other cases of a similar nature the course in this one was relatively chronic, death not taking place till 9 days after the onset of the symptoms.

Apart from the mode of development of the disease, the case presented several manifestations of clinical interest. The onset of constipation was gradual; it only became absolute a few hours before admission, but continued so until the death of the patient 2 days later. There was a total absence of blood in the stools throughout the illness, and haematemesis did not occur till 48 hours before death.

A physical sign of some importance but of considerable rarity was present in this case; a localised area of resistance, which proved under the anaesthetic to be a definite mass, was felt in the left iliac region. The mass consisted of the infarcted coil and its mesentery which was thickened by oedema.

CASE IV

Atheromatous 'ulcer' in the aorta close to the origin of the superior mesenteric artery; 'ulcer' covered by thrombus extending into and completely occluding trunk of superior mesenteric. Haemorrhagic infarction of part of the intestine. Under the care of Dr H. BATTY SHAW.

HISTORY. The patient was admitted to University College Hospital on Sun., Aug. 16th, 1903, at 5 p.m. complaining of malaise and vomiting of several days' duration, constipation (no stool passed) since Tues. 11th, and haematemesis for the first time on Sun. 16th.

PRESENT STATE. The abdomen is very distended and tender in its lower half; no visible peristalsis is present. Pt. keeps on vomiting blood about every half-hour, the vomited matter later in the day becoming stereoraceous in odour.

The pulse is extremely weak, 124 per min. The heart-sounds can scarcely be heard. The temperature is 98·4, and the respirations 36 per min.

After an enema blood-clot only came away. The urine contains blood and about 50 % of albumin.

7 p.m. Operation was considered but was decided against on account of the bad general condition. Glycerine and belladonna fomentations were applied to the abdomen, and the following mixture given by the mouth:—

Ac. hydrocyan. dil. m. iii.

Tr. opii. m. x.

Aq. chloroformi ad $\frac{1}{2}$ oz. every 4 hours.

The vomit became gradually more stercoraceous, and pt. became almost pulseless. At 7 p.m. pt. was given a subcutaneous saline infusion.

At 10 p.m., T. 100·4, P. 140, and R. 40.

Aug. 17th. 3 a.m. Pt. was given a second subcutaneous infusion. T. 99·4, P. 108, and R. 44.

9.30 a.m. Pt. died after a convulsion.

AUTOPSY. A quantity of free blood was found in the peritoneal cavity, but there was no sign of peritonitis.

The intestines were much distended, and the last 5 ft. of the ileum, together with the caecum, and part of the ascending colon were nearly gangrenous; the mucosa of the affected intestine contained numerous ecchymoses.

At the junction of the superior mesenteric artery and the aorta there was an atheromatous 'ulcer,' and upon this a clot had formed occluding the superior mesenteric; the clot was firmly attached, and extended along the artery for about 3 ins.

Both kidneys were of the 'large white' variety.

The liver shewed fatty degeneration.

The spleen was normal.

The lungs shewed congestion of the bases.

The heart was very fatty; the right side was dilated; there were no other changes.

The cause of the thrombosis was almost certainly atheroma of the aorta over the origin of the superior mesenteric.

CONCLUSIONS.

There are two main points of interest in this case:—(1) Clinical. The occurrence of constipation at the time of onset is rare in this disease. Unfortunately no data are available regarding the state of the bowels before the onset in this case, but at all events constipation was a prominent symptom at a very early stage. The other clinical manifestations were such as may be expected in this disease. (2) Pathological. As will be seen later (*vide* 'Classification; arterial') the causal lesion found in this case, viz., an atheromatous 'ulcer' near the origin of the superior mesenteric artery, is very rare. A thrombus had formed upon the 'ulcer' and had spread into the trunk of the superior mesenteric.

It appears probable that the occlusion was not completed at once, as the thrombus found at the autopsy was firmly adherent to the arterial wall.

The most likely explanation seems to be that the superior mesenteric had been partly occluded by the thrombus for some length of time before death, and that the acute symptoms supervened only when absolute obstruction of the artery occurred.

CASE V

Acute appendicitis; removal of the appendix; 'primary' mesenteric venous thrombosis; local peritonitis; recovery. Under the care of Mr WILFRED TROTTER.

HISTORY. The patient was admitted (after walking up to the ward) for 'acute appendicitis' upon the recommendation of his doctor, and gave the following history:

Sudden sharp attack in the lower abdomen on April 15th; he vomited once. The pain had persisted but had been less during the last few days; it had not been more marked on the right side. No fever during illness and no further vomiting. Bowels had been open daily with medicine. No pain on micturition. He had had two previous attacks of pain in the lower abdomen, but they had not been so bad as the present one. He did not work in lead.

SUPPLEMENTARY NOTE. A few days before he had purchased some laudanum for his pain.

PRESENT STATE (April 23, 1911). Pt. is an anaemic weak-looking man of 29; much wasted; cheeks sunken. The pulse is 82 per min., the temperature 98, and the respiration 24 per min. The tongue is thickly furred, but moist. He is in no pain, and lies comfortably in bed. He has not been given any morphia.

Cardio-vascular system is normal.

Respiratory system is normal, save for some harsh breath sounds at the right apex.

Pupils are of moderate size and react to light and accommodation.

Urine is normal.

Abdomen. No general distension, but slight fulness in right lower abdomen. Movements good and symmetrical. No impairment of resonance anywhere, and no tenderness. Some increased resistance over the right lower abdomen.

A non-tender mass can be felt just to right of umbilicus, and another in region of sigmoid.

Per rectum, a non-tender body feeling like the appendix can be felt hanging over the brim of the pelvis on the right side.

Pt. was given an enema; constipated result; nothing abnormal present.

8 p.m. T. 100, P. 98, and R. 24. No pain and no tenderness anywhere.

OPERATION (11 p.m.). Mr Wilfred Trotter. Scopolamine, morphine, atropine, and 'open' ether. Abdomen opened by the usual muscle-splitting appendix incision. No free fluid. A mass, apparently composed of enlarged glands—the size of a small tangerine orange—was felt in the angle between ileum and colon, in the root of the mesentery; no fluctuation obtained in this swelling. Caecum was lifted forwards and the appendix found; it was acutely inflamed in its distal $\frac{1}{3}$ and kinked about the middle, the kink being behind the caecum and the distal $\frac{1}{3}$ hanging down over the brim of the pelvis. No local peritonitis but some congestion on surface of caecum. No local abscess. Appendix was firmly adherent and bleeding was rather profuse. Appendix was freed and removed by clamp and cautery, and the

stump invaginated. Abdomen was closed in layers with catgut. No drainage.

Appendix on section shewed a little pus in the distal end, and early purulent infiltration of mucosa in same region. There was thickening at the site of kink.

Apl. 24th. Very comfortable.

Apl. 25th. Some hiccough. No undue tenderness anywhere.

Apl. 26th. Pt. had a rigor to-day for a quarter of an hour, and T. rose to 103·8. P. 120. Venous thrombosis now suspected in view of mass felt at operation. R. 32. Bowels open to-day; no blood in stool, which was constipated. No change in local signs.

Apl. 27th. Pt. was given a stock vaccine of *Streptococcus* and *B. Coli* (1 milln. *Strept.* and 1 milln. *B. Coli*). T. 100. Tenderness and rigidity in right lower abdomen. Wound healthy.

Apl. 28th. Pt. not so well. Wound dressed. No undue induration in wound, but lower abdomen is practically motionless and somewhat tender; no mass felt. Hiccough is troublesome. Nothing definite felt per rectum.

8 p.m. T. 98·8, P. 88. Pt. has vomited bile-stained fluid twice this evening. Bowels confined since Apl. 26.

SECOND OPERATION. 11 p.m. Mr Morrison Davies. Spinal anaesthesia.

Incision in right iliac region opened up. The mesentery of the small intestine for the last 10 ins. was found to be considerably swollen and indurated, the induration extending to its root; it reached the intestine about 8 ins. from the ileo-caecal junction, and here the ileum for a distance of about 4 ins. was of a bluish congested appearance. It was not paralysed, since strong peristaltic waves were seen passing along it. No peritonitis. The mass felt at the previous operation now found to be due to thickening of the mesentery. Abdominal wall stitched up.

Apl. 29th. T. 102·4, P. 104. Pt. has not vomited since operation. Vaccine = 5 m. *Strept.* and 5 m. *B. Coli*.

Apl. 30th. Enema given this evening with good result; no blood; pt. has passed flatus.

May 3rd. T. has risen again. Great difficulty in moving bowels; no blood or mucus in stools. No pain. Pulse remains

good. Pt. is getting very much thinner. Vaccine = 10 m. of each.

May 7th. T. lower. Enemata necessary for bowels. Lower abdomen distended, and does not move very well. No dulness in flanks. Appetite bad. Vomited once.

May 8th. Vomited again to-day; bile-stained material. Per rectum: anterior rectal wall bulging downwards; tenderness high up on left side.

May 9th. Vomited 50 oz. Lower abdomen motionless and rather tender. Some dulness in right flank. T. normal, P. 120.

THIRD OPERATION. Mr Trotter. Spinal anaesthesia. Incision reopened; it had healed perfectly. Caecum found adherent to scar. On opening peritoneal cavity a large quantity of purulent inoffensive fluid was evacuated. In the floor of the abscess were coils of intestine, and at the very bottom, lying in the pelvis, was the thrombosed loop of intestine. This was very adherent and was separated with great difficulty. There was active peristalsis in the small intestine forming the wall of the abscess. The thrombosed portion of mesentery felt much thickened, and was more extensive than that found at the previous operation. The affected coil of bowel was very much congested and covered with lymph.

The pus was swabbed away, and the affected coil of intestine was freed from the floor of the pelvis. Wound closed completely (catgut sutures for deep layers and silkworm gut for skin). Pt. stood operation well.

11 p.m. P. good, 110. Continuous rectal saline infusion. Normal saline subcutaneous infusion (1 pint). Hiccough continues.

May 11th. T. normal. All pain gone; pt. feels much better; no more vomiting. Vaccine = 10 m. of each (B. Coli and Strept.). Bowels open twice last night; no blood in stools.

May 13th. Dressed; abdomen distended and tender in lower part.

May 14th. T. subnormal. Lower abdomen still distended. Pt. has hiccough badly and pain in abdomen. No vomiting. P. 108. Bowels open well and frequently.

FOURTH OPERATION. 1 p.m. Mr Trotter. Spinal anaesthesia.

Incision reopened. Abscess-cavity was full of purulent inoffensive fluid; cavity smaller than at last operation. Thrombosed coil again lying adherent to pelvis; freed. A considerable fresh abscess was opened in bottom of pelvis, in front of rectum. Its contents consisted of thick pus; swabbed dry, and a large drainage tube inserted.

Abdominal wound partly closed with catgut and with silkworm gut.

May 15th. Dressed. Complete relief of symptoms has followed the operation.

May 16th. Tube removed and replaced by a smaller one.

May 21st. Daily dressing. T. rising again. No pain in abdomen.

May 24th. T. 101·8, P. 128.

May 25th. Larger tube inserted to-day. Abscess-cavity is being washed out daily with hydrogen peroxide and iodine.

Pt. is looking much better and taking food well.

May 27th. Sinus is closing well. T. nearly normal. Still difficulty in opening bowels; stools normal.

June 6th. Pt. complains of pain in appendix region; bowels rather constipated. T. normal. Discharge less, but possibility of accumulation shut off from drainage considered.

FIFTH OPERATION. Mr Trotter. Spinal anaesthesia.

Wound enlarged and sinus explored with finger. No collection of pus discovered. Drainage tube inserted. Symptoms considered to be entirely due to adhesions.

Some faintness and nausea after operation, possibly due to stovaine.

June 11th. T. normal. Pt. quite comfortable. Nausea and faintness have completely vanished. Tube shortened.

June 12th. T. 99·8. Pt. feels well.

July 4th. Uninterrupted progress. Slight discharge from wound still. No pain. Pulse quiet. T. normal. Pt. is going to convalescent home to-day.

July 29th. Pt. returned from convalescent home looking very well. Slight subcutaneous abscess burst spontaneously while he was away; wound has now healed completely. Pt. has gained weight very rapidly.

CONCLUSIONS.

This case presents many points of interest from the standpoint of the history, the course, and the treatment.

(1) History. The patient had had three attacks of appendicitis, the last of which was acute. After the last attack the symptoms shewed marked remission for several days, and at the time of admission to hospital the pain had ceased completely. There had been no fever and no rigors.

(2) Course. There were never any of the fulminating symptoms such as are to be expected in cases of mesenteric vascular occlusion; the symptoms and signs subsequently to the first operation were those of acute intestinal obstruction. Fever, distension, hiccough, rapid pulse, and vomiting were recurring manifestations, but pain was never very acute. The relief after each operation was a striking feature of the case, but the symptoms returned, repeatedly. The coil of affected intestine was never paralysed though it was much congested and thickened together with its mesentery.

(3) Treatment. After the initial operation for the removal of the appendix, the treatment consisted for the most part of freeing the affected coil from adhesions which bound it to the floor of the pelvis. The beneficial results of each operation were doubtless due to several factors. Of these the most important probably was the relief of the affected coil from adhesions and impaction in the pelvis. This obstruction, had it been unrelieved, would undoubtedly have been fatal. Secondly, it seems likely that the peritonitis would have developed into a more formidable complication. While, thirdly, the facts seem to suggest that the operations may have had some effect in preventing the vascular lesion from progressing to actual infarction.

This case is of importance too as an illustration of the value of spinal anaesthesia. Within the space of 48 days the abdomen was explored no less than 5 times. If any of the older methods of anaesthesia had been employed, cardiac or pulmonary complications might have led to fatal consequences. With the exception of the first operation in which ether (by the 'open' method), following scopolamine, morphine and atropine, was employed, all the operations were performed under spinal anaesthesia (stovaine).

CASE VI

Aneurism of the abdominal aorta, ruptured. Old, complete closure of superior mesenteric artery and coeliac axis, and partial closure of inferior mesenteric artery. Establishment of complete collateral circulation. No infarction or morbid changes in stomach or intestine. Under the care of Dr ALEXANDER MORISON.

Feb. 27th, 1911. Pt. was admitted to the Great Northern Central Hospital complaining of:

Pain across the back, and a throbbing lump in the left side of the abdomen.

HISTORY. Two years ago it was noticed that there was something the matter with his heart. He had previously suffered a good deal with indigestion and biliousness. Pt. first noticed the lump 18 or 20 months ago; this gradually became bigger, painful and tender. There has been no pain in it for the last 3 weeks, and the lump has grown much harder. Pt. has been in bed during the last 5 weeks. The pain across the back keeps him awake at night.

PREVIOUS HISTORY. Dysentery 12 years ago; ill 7 months.

FAMILY HISTORY. Pt.'s father died of aneurism.

PRESENT STATE, Feb. 27th, 1911. Pt. is a fairly healthy looking man of 46; rather pale; fairly well covered. Tongue moist, slightly furred. Teeth: many artificial; others fairly good. Eyes: movements normal; pupils react to light and accommodation; at times left pupil appears larger than right. Temperature 98·8, pulse 96 to 104 per min.

Chest. In left hypochondrium pushing 7th and 8th ribs forward rhythmically and extending below costal margin nearly down to umbilicus is a pulsating swelling¹, smooth, round, not attached to skin; outline of swelling fairly definite; about size of fist; surface hard; dull on percussion. No murmur heard over it, but there is over abdominal aorta below it. Pulsation of swelling synchronous with heart-beat. Not tender, but painful at times. Tumour is fixed in relation to surrounding parts, but moves slightly on respiration.

¹ See Figs. 3 and 4.

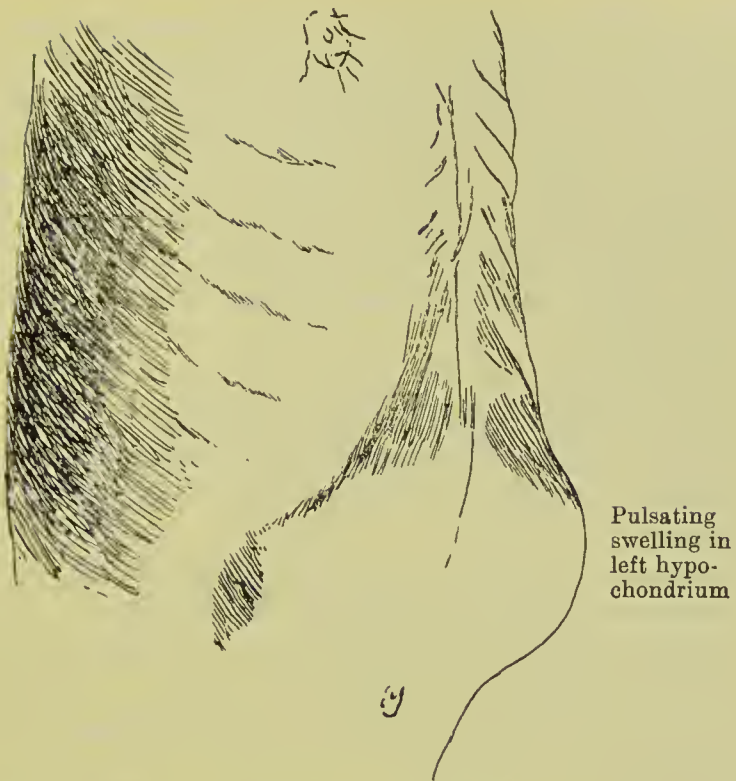


Fig. 3. Aneurism of aorta, side view. April 19th, 1911.

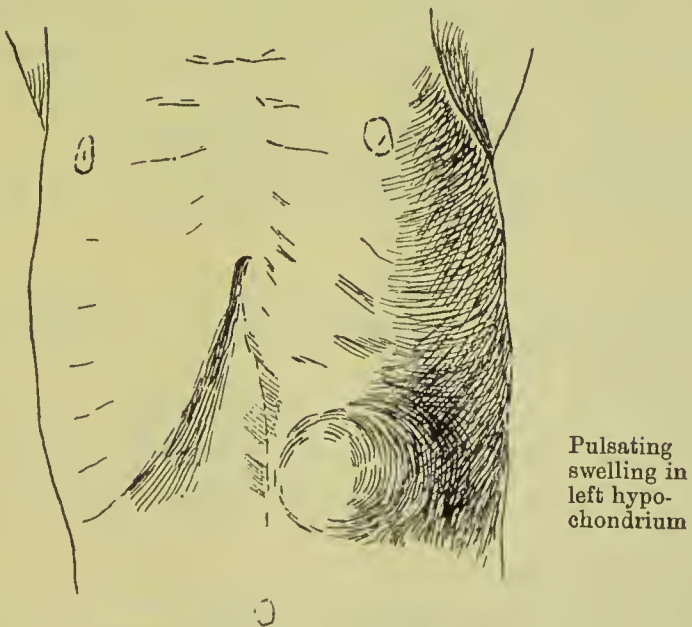


Fig. 4. Aneurism of aorta, front view. April 19th, 1911.

Heart. Visible apex beat in 5th left intercostal space 3 ins. from mid sternum, where there is a triple rhythm (*bruit de galop*). Along left edge of sternum as high as 2nd rib is a diastolic bruit which is also audible over 2nd right cartilage; highest note is over 4th left cartilage. The right percussion border of heart is at left edge of sternum. Area of cardiac dulness is 4 ins. $\times$ 4½ ins.

Lungs normal.

Liver. Upper limit of dulness 6th rib. Vertical measurement of dulness 4½ ins.

No albuminuria.

Pt. is being kept in bed. Diet, chicken jelly.

Potassium iodide gr. 5 is being given thrice daily.

Feb. 27th. Dose of Pot. iod. increased to gr. 10. Diet, fish and chicken.

March 6th. Aneurism seems harder; no pain. Blood-pressure: right brachial artery 185 mm. hg., left brachial 170.

March 8th. Hypodermic injection of 1 % gelatine in normal saline solution (3 oz.) into axilla.

March 13th. Apex beat 5th space in nipple-line 3½ ins. from mid-sternum.

March 17th. Gelatine injection as before.

March 20th. Dose of Pot. iod. increased to gr. 15. Ribs and cartilages 6, 7 and 8 powerfully heaved by aneurism during systole.

March 22nd. Wassermann's reaction positive.

March 23rd. Gelatine injection as before.

March 26th. Dose of Pot. iod. increased to gr. 20 (thrice daily).

March 31st. Gelatine injection.

Apl. 7th. Gelatine injection omitted. Pt. continues to feel quite comfortable.

Apl. 15th. Allowed to get up a little; feels much better

Apl. 22nd. Slight attack of haematemesis (½ oz.) accompanied by a great deal of pain in the epigastrium. Morphia gr. 1/8. Calcium lactate gr. 60. Rectal salines and nutrient enemata. Mouth feeding stopped.

Apl. 23rd. Ice bag applied to epigastrium for aching pain. No further haematemesis. No melaena. T. 100.

Apl. 25th. Area of pulsation appears to be increasing upwards. Pt. still has aching pain. Kept in bed since 22nd. Diet, Brand's essence by the mouth. Appetite very poor.

Apl. 27th. Diet, chicken jelly and milk in small quantities frequently.

May 1st. Pt. not so well last night; discomfort in evening, which he attributes to swallowing some milk a little too quickly. At 12 p.m. he was suddenly seized with extremely acute pain in the epigastric region. When seen a few minutes later he was rolling about in agony, clutching at the bed-clothes; he was semi-conscious apparently; the pupils were widely dilated, the face extremely pale and covered with cold sweat, the extremities very cold, and the pulse just palpable (at least 140 per min.); all signs of severe internal haemorrhage. Aneurism not pulsating so forcibly as before. Shifting dulness in upper abdomen, increasing during the next half hour. Morphia gr. $\frac{1}{4}$ given.

The pulse became gradually feebler; dyspnoea 12.30 a.m. till death 12.40 a.m. At time of death the abdominal tumour dropped back and the abdomen and lower chest appeared symmetrical.

Throughout the illness the temperature never rose to 100 with the single exception of Apl. 22nd, i.e., after the attack of haematemesis.

AUTOPSY (Partial). The body is that of a poorly nourished man. Lungs slightly congested. Pericardium natural.

Heart. Uniform slight enlargement; pulmonary, tricuspid and mitral valves natural; aortic segments a little thickened and irregular; wall of left ventricle hypertrophied and cavity a little dilated.

Aorta thickened; shews many atheromatous patches along the arch; from the arch to the diaphragm it is not much affected by atheroma. A large aneurism springs from the aorta just below the diaphragm. The mouth of the sac is very wide and involves the whole anterior part of the aortic wall; posteriorly there is also some bulging. The tissues about the sac are thick and fibrous, and adhesions bind the aneurism to the stomach. The first lumbar vertebra is eroded by the aneurism to a depth of about $\frac{1}{3}$ in. and forms part of its wall. A layer of laminated friable clot lines the wall of the sac, the whole being about $\frac{1}{3}$ in.

thick. There is a slit in the upper left-hand aspect of the aneurism representing the position of the rupture, but it is not possible to define it exactly owing to the large amount of blood-clot present. The blood-clot is soft and red and extends under the peritoneum to the pelvis below, also under the mesentery to the right of the spine, and forwards it has separated the peritoneum from the cardiac end of the stomach. The left kidney is surrounded by clot to a depth of over one inch. Black gelatinous fibrinous clot covers the red clot in places. A little blood-stained serum is present in the peritoneal cavity. The abdominal aorta and iliac arteries are markedly atheromatous.

The liver is pale and very nodular on the surface; it is about the natural size.

The kidneys are pale and granular; there is no sign of infarction.

The spleen is natural.

The suprarenals and pancreas are natural.

The stomach and intestines are normal; they contain no blood, and there are no macroscopic signs of haemorrhages, old or recent.

EXAMINATION OF ANEURISM after preservation in formalin¹.

The whole aneurism measures $4\frac{1}{2}$ ins. from its proximal to its distal extremity, the latter being $2\frac{1}{2}$ ins. above the space between the common iliac arteries. The antero-posterior measurement is 5 ins.

The sac has been formed at the expense mainly of the left anterior wall of the aorta between the two crura of the diaphragm, and consists essentially of two parts:

(1) The main chamber which is roughly fusiform, and measures about $3\frac{1}{2}$ ins. from side to side by $4\frac{1}{2}$ ins. from above downwards; and

(2) A dome-shaped prominence rising $2\frac{1}{2}$ ins. to $2\frac{3}{4}$ ins. above the first part, and extending almost directly forwards so as to press the costal cartilages forwards during life. This dome measures about $4\frac{1}{2}$ ins. from side to side, and about 2 ins. from above downwards.

The aneurism after dissection is found to have given way at

¹ See Fig. 5. The specimen has since been presented to the Museum of the Royal College of Surgeons, London.

two separate spots, both situated immediately above the dome described above.

The left-hand opening runs longitudinally, and measures about $1\frac{1}{2}$ in. at the outer surface of the sac. It is full of hard, almost black blood-clot.

The right-hand rupture is about $\frac{3}{4}$ in. long, and runs almost transversely, the left-hand extremity being situated $\frac{1}{2}$ in. from the longitudinal slit.

The cavity of the sac is smooth except where the wall has been ruptured; here the laminated clot forming the main thickness



Fig. 5. Aneurism of abdominal aorta seen from the right side.

- | | |
|--|--|
| 1. Aorta passing behind diaphragm. | 6. Right renal artery. |
| 2. Portion of right crus of diaphragm. | 7. Inferior mesenteric artery. |
| 3. Main part of aneurism. | 8. Dome-shaped anterior part of aneurism. |
| 4. Coeliac axis. | 9. One of the points of rupture of aneurism. |
| 5. Superior mesenteric artery. | 10. Aorta below aneurism. |

of the wall has been split up, and is very friable. For this reason it is impossible to make a thorough examination of the interior of the sac without destroying the specimen.

The fusiform part of the aneurism varies from $\frac{1}{16}$ in. to $\frac{1}{4}$ in. in thickness, while the wall of the dome becomes thicker as the apex is approached. The maximum thickness of the latter is $\frac{1}{2}$ in.

ARTERIES ARISING FROM THE SAC. Half an inch below and to the right of the distal margin of the foot of the dome-shaped prominence lies the orifice of the coeliac axis. A bristle cannot be passed through it into the aneurism, and the orifice of the vessel on the inner surface of the sac is represented merely by a shallow depression or dimple.

The origin of the superior mesenteric artery is situated $\frac{1}{2}$ in. below the coeliac axis. This also is completely occluded (*vide* Plate II) by old thrombus about .5 cm. long, and by contraction of the orifice.

One inch to the right of the superior mesenteric artery is the right renal artery, also completely occluded.

The inferior mesenteric artery is permeable though the lumen is narrowed, and arises $1\frac{1}{2}$ in. from the foot of the distal side of the dome.

The left renal artery unfortunately is not to be found; it must have been torn away while the aneurism was being separated from the vertebral column to which it was firmly attached.

Since at the time of the autopsy it was never suspected that the vessels mentioned above were occluded, no effort was made to trace the vessels that supplied the collateral circulation.

HISTOLOGICAL EXAMINATION¹.

Longitudinal sections were made from above downwards through the origin of the superior mesenteric artery. When the artery was cut open, a grey, firm, fibrous clot was found, partly adherent to the intima. Unfortunately, during the preparation of the specimen this thrombus, which measured about .5 cm. in length, became detached, it is therefore not represented in the appended drawing.

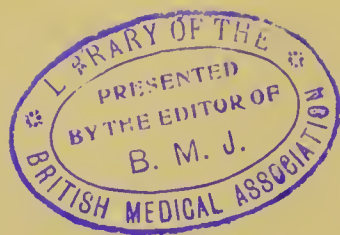
On the proximal side of the thrombus the orifice of the artery is completely closed by fibrous tissue which has led to kinking of the intima, so that it is difficult to be sure of the actual site of the original opening from the aorta. The fibrous tissue is arranged in bundles which run in different directions and give an irregular texture to this part of the wall of the aorta. The intima of the portion of the aorta in the section varies considerably in thickness,

¹ See Fig. 5.



Hubert H. H. H.

- A=intima of superior mesenteric artery.
B=intima of aorta.
C=fibrous tissue bundles running in different directions.
D=lumen of superior mesenteric artery.



and in one place it is entirely absent; here it has been replaced by fibrous tissue. There is considerable thickening of the intima of the superior mesenteric as it passes through the wall of the aorta.

No histological examination of the inferior mesenteric artery was made as it appeared to have undergone the same changes as the superior, though the process had not advanced so far.

CONCLUSIONS.

This case is of exceptional interest in that it presented complete occlusion of the superior mesenteric artery and coeliac axis, and very marked narrowing of the lumen of the inferior mesenteric without the patient having manifested any of the symptoms one expects to find associated with obstruction of these vessels. The terminal attack of agonising pain was in all probability caused by the stripping of the parietal peritoneum off the posterior abdominal wall by haemorrhage after rupture of the aneurism.

In seeking for an explanation for the total absence of gastrointestinal lesions after closure of the arteries of supply, we have to bear in mind two important facts. In the first place, a pulsating tumour had been noticed in the left hypochondrium for nearly two years before death, and in all probability the aneurism had begun to develop some months earlier. The period therefore during which the origins of the affected arteries were becoming constricted was evidently about 2 years; it may be supposed that during this interval the collateral arterial channels were gradually becoming functionally active.

In the second place, the patient had been taking a specially restricted diet for a considerable time before death on account of the aneurism. The amount of work required of the intestine, and the consequent need for arterial blood by that viscus were therefore much less than the normal.

These two circumstances would explain the absence of any morbid-anatomical changes in the areas of supply of the occluded vessels.

CASE VII

Atheromatous 'ulcer' in the aorta, close to the origin of the superior mesenteric artery; 'ulcer' covered by thrombus; embolism of superior mesenteric artery. Haemorrhagic infarction of part of jejunum. The notes of this case under treatment at the London County Lunatic Asylum have been placed at the disposal of the writer through the courtesy of Dr J. B. HOWELL.

HISTORY. Rheumatic fever in 1901; heart disease followed. Epileptic.

April 20th, 1909. Pt. has mitral regurgitation. Apical systolic murmur.

Dec. 14th, 1911. Heart somewhat enlarged; impulse slapping and irregular; presystolic murmur at apex; 1st sound short and sharp; 2nd present.

March 10th, 1912. Pt. complained yesterday of some pain in the abdomen. Bowels not open. No physical signs. She is always very constipated, and says that periodically she has attacks of colic which pass off in a few days.

PRESENT STATE. March 10th, 1912. Female of 57. Pain persists, and there is some flatulent distension of the abdomen. No organ or mass to be felt. No motion but flatus has been passed.

March 11th. Very much worse; abdominal facies; pain remains; abdomen more distended; no motion and no result from turpentine enema. Indefinite mass felt in right iliac fossa; mass pits on pressure (? faeces). Pt. has vomited once.

OPERATION. Incision along outer margin of right rectus; caecum and appendix presented, both healthy; black gangrenous gut next brought out. The vessels of this part were thrombosed, as also the vessels of a wedge of mesentery. About 4 ft. of this gut (jejunum) resected with wedge of mesentery, and lateral anastomosis effected.

March 12th. Pt. went steadily downhill, and died at 3 p.m. to-day.

AUTOPSY. Heart. 335 grms. Rt. ventricle normal; auricles filled with decolourised clot which is fairly firmly attached to the

wall; valves healthy; lt. ventricle hypertrophied; marked mitral stenosis; cusps thickened and calcareous; no vegetations.

Aorta shews slight early atheroma, and there is an ulcerated patch just above the opening of the superior mesenteric artery; there is an organised thrombus resting upon the ulcer, and it is evidently from this clot that the mesenteric embolus originated.

Coronary arteries healthy.

Peritoneum. Some plastic peritonitis with a few flakes of lymph; no free blood nor bowel-contents.

Small intestine. About 18 ins. from duodenum 4 ft. of gut has been removed, and a lateral anastomosis effected; the cut ends are perfectly closed, and allow no leakage; the anastomosis is firm, and the lumen free and adequate.

Microscopically the thrombosed vessels of the mesentery shewed no morbid changes dating previously to the thrombosis.

CONCLUSIONS.

This case should be considered in conjunction with case No. 4 (original), the causal lesion in the two instances being almost precisely the same.

This case, like case No. 4, presented an atheromatous 'ulcer' in the aorta close to the origin of the superior mesenteric artery; a sessile thrombus had formed upon the 'ulcer,' but instead of the thrombus spreading by direct continuity into the superior mesenteric, this vessel was occluded by a portion which had become broken off.

With regard to the clinical manifestations it is difficult to say how much importance is to be attached to the constipation, as the patient had been under restraint for epilepsy and had been habitually constipated; further, she had been subject to severe attacks of colicky abdominal pains lasting for days at a time.

The course of the disease was acute, the patient dying 3 days after the onset of severe symptoms.

SECTION II

CLASSIFICATION OF MESENTERIC VASCULAR OCCLUSIONS FROM THE STANDPOINT OF MORBID ANATOMY

IN the present state of our knowledge it is impossible to differentiate clinically between lesions arising from occlusions of the arteries and those arising from occlusion of the veins in the mesentery. On the other hand, there does appear to be some relationship between the severity of the clinical manifestations and the degree of the pathological process. That is to say, in those cases where the vascular lesion has led to infarction of the intestine the signs and symptoms have been more intense, as a rule, than in those where compensation in the circulation has taken place. In certain cases of minor severity that have not proved fatal, or have not required operative interference, it has been possible to suspect the nature of the pathological lesion only from the coexistence of a known possible cause: this may be an endocarditis giving rise to embolic manifestations elsewhere, a cirrhosis of the liver, an appendicitis or other septic condition.

One may say with safety that the clinical phenomena associated with a mesenteric arterial or venous occlusion, though when taken in conjunction with associated morbid conditions they may be at times sufficient to suggest either lesion, practically never present themselves with such clearness as to warrant a certain diagnosis. It will be convenient therefore for purposes of discussion to classify the cases of mesenteric vascular occlusion collected in this paper under the following headings:—

- (a) Cases resulting from venous obstruction,
- (b) Cases resulting from arterial obstruction, and

- (c) Cases in which both mesenteric artery and vein have been occluded simultaneously.

If we compare the relative frequency of arterial and venous occlusions in the mesentery, we find that the disproportion in the number of cases due to the two conditions is not so great as has generally been supposed. Thus out of about 360 cases in this series about 41 % were due to venous obstructions, 53 % to arterial, and 6 % to simultaneous arterial and venous obstructions¹. It is possible that the disproportion is even less than is indicated by the figures, since the clinical picture which leads one to suspect arterial embolism is rather better defined than in the case of venous thrombosis. For example, when a patient who is known to have a valvular cardiac lesion suddenly develops acute abdominal pains, diarrhoea and vomiting, with blood in the stools or in the vomited matter, and distension, the whole clinical picture is very suggestive of mesenteric embolism. Such cases do not always prove fatal, and the diagnosis remains unconfirmed. On the other hand, the associated conditions in cases of venous thrombosis are not usually so apparent.

(a) MESENTERIC VENOUS OCCLUSION.

The cases of venous obstruction that give rise to grave clinical manifestations are almost always those presenting lesions of the superior mesenteric or portal vein. Obliteration of the inferior mesenteric vein alone, without simultaneous involvement of the superior mesenteric or portal vein, practically never leads to infarction², the reason being that the inferior mesenteric vein forms richer anastomoses with the systemic venous system relatively to the amount of blood to be carried away than does the superior, rendering possible the establishment of a more efficient collateral circulation. For this reason, if for no other, thrombosis of the inferior mesenteric vein *per se* is a condition of incomparably smaller clinical significance than that of the superior mesenteric.

The venous circulation in the intestines can be carried on even if a large part of the mesenteric venous system has undergone thrombosis. So long as tributaries that are free from clot open

¹ These figures for the sake of comparison include all probable cases.

² Köster's case is exceptional in that infarction occurred.

into the main trunk above the highest limit of the clot, and the anastomosing arcades and their radicles leading from the intestine remain patent, the intestine may remain free from infarction¹. The great danger in such cases lies in the extension of the clot peripherally into the anastomosing arcades and their radicles: even then the infarct is often limited to the segment corresponding merely with the occluding radicles. This fact was well illustrated by Bradford's case and No. 3 of the original cases reported above. In both of these cases a very extensive thrombosis obtained, but infarction was limited to that sector of mesentery and intestine in which the anastomosing arcades and their radicles had also undergone thrombosis.

It may be noted here that Mauclaire and Jacoulet, while discussing the extent and significance of thrombosis in the mesentery, state that the clot rarely fills the lumen of the vein, and usually but lines (*'tapisse'*) the walls. The evidence brought forward for such a general statement appears to be inadequate, and it has not been possible to find any support for it in the works of other authors. Maygrier and Letulle, it is true, describe the formation of a thrombus from without inwards in their case of thrombophlebitis following puerperal septicaemia: Frerichs² also says that the periphery of the clot was laid down in layers in his second case of portal thrombosis. In both these cases however there was no question of the incomplete filling of the lumen.

¹ There is abundant evidence to prove that a complete collateral venous circulation may be established if the superior mesenteric or even the portal vein is occluded. The superior mesenteric was tied near its termination in two cases (Wilms, Mayo Robson): in both cases the vein had been lacerated, and both the patients recovered. The venous return after the ligature of the mesenteric probably took place through the right gastro-epiploic and upper jejunal veins, and the colic and inferior mesenteric, all of which had to adapt themselves suddenly to the change in the circulation. Brewer ligatured the portal vein with success in a case of hydatid of the liver: in this case the vein had already become reduced in size, and a collateral circulation therefore to some extent been established. Numerous cases of portal thrombosis are on record in which the patients survived a considerable time, e.g. those reported by Pick, Umber (both reviewed by de Jong), Sprengel, de Jong; also three cases in Jackson, Porter and Quinby's series, and two from which specimens are preserved in St Bartholomew's Hospital Museum. De Jong emphasises the importance of the veins in the gastro-hepatic omentum, and in the suspensory ligaments of the liver, in his discussion upon the collateral circulation in cases of this sort.

² Borrmann, *loc. cit.*, S. 288.

Cases of thrombosis of the superior mesenteric vein may be divided into two main groups according to their aetiology¹:—

(1) Primary, in which the process started in the superior mesenteric vein itself, and

(2) Secondary, in which as a terminal incident the superior mesenteric vein became involved in a descending thrombosis originating in the portal vein, or its intra-hepatic branches.

In view of the greater frequency of secondary thrombosis it will be convenient to deal with group No. 2 first. Incidentally, we shall have to consider briefly the subject of portal thrombosis first of all, in order to understand the pathology of descending mesenteric thrombosis.

Portal thrombosis may be caused by a condition of sepsis in any part of the alimentary tract or other viscus which drains into the portal system. Organisms from a septic focus may initiate a thrombosis in the portal vein though there may be no thrombi in the veins at or near the septic focus itself. Thus a pylephlebitis² or a thrombosis of the portal vein may follow an appendicitis, apart from thrombosis in the appendicular veins.

The commonest predisposing cause of portal thrombosis is pressure upon the vein from without. This may be exerted by cicatricial contraction of the liver capsule about the portal fissure as a result of syphilis³ or alcoholic cirrhosis⁴, by primary or secondary carcinoma⁵ of the liver, by metastatic growth outside the liver⁶, by gumma of the liver⁷, by gall-stones⁸, by enlarged lymphatic glands about the portal fissure⁹, and finally by fibrotic adhesions in the same region¹⁰.

Lissauer, who investigated very thoroughly the subject of

¹ A certain amount of overlapping is unfortunately unavoidable in this method of classification.

² Polya collected 23 cases of pylephlebitis out of 469 fatal cases of appendicitis.

³ Bülow (reviewed by Borrmann). ⁴ Aehard (ditto).

⁵ Lissauer. ⁶ De Jong reports one original case.

⁷ Jastrowitz, and Bülow (both cases reviewed by Borrmann).

⁸ Lissauer states that gall-stones may produce portal thrombosis by pressure alone, or by pressure and sepsis acting together.

⁹ Glands about the portal fissure were held responsible in 13 out of Lissauer's series of 68 cases.

¹⁰ Frerichs (reviewed by Borrmann), and London Hospital P. M. Records, 1908, No. 1144, and 1907, No. 565.

portal thrombosis, found that morbid conditions of the liver (most of them acting by pressure upon the vein) were responsible for the process in 47 % of a series of 68 cases. His analysis of the causal lesions is quoted below:—

Atrophic cirrhosis	6 cases	9 %
Gumma and syphilitic perihepatitis	7 „	10 %
Primary cancer	2 „	3 %
Secondary cancer	2 „	3 %
Primary cancer of the gall-bladder	6 „	9 %
Gall-stones	9 „	13 %
		47 %

Borrmann and de Jong ascribe great importance to syphilitic changes of a sclerosing nature pressing upon and frequently involving the wall of the portal vein, or its intra-hepatic branches. Advanced fibrosis of the portal vein is probably a favourable condition for the development of thrombosis on account of its usual association with changes in the intima.

The factors, in Borrmann's opinion, that are of especial significance in the causation of fibrosis are old age, high blood-pressure, intoxications and infectious diseases—particularly alcohol and syphilis.

Thrombosis has been associated in a few cases with definite calcareous changes in the portal vein¹.

Again, portal thrombosis may take place as the result of a grafting, so to speak, of a simple thrombus upon a malignant one produced by the invasion of a portal tributary by growth in the stomach or pancreas².

Finally, four cases of portal thrombosis following splenectomy have been reported. In one³ total infarction of the small intestine resulted; in a second⁴ the process was ascribed to double pneumonia; while in the third⁵ and fourth⁵ the diagnosis of the portal condition was open to some doubt.

¹ Borrmann reviews 14 cases of this description, and Rolleston reports one.

² Rolleston, Askanazy.

³ Jonnesco (reviewed by Maclaure and Jacoulet).

⁴ Dclatour.

⁵ Lieblein.

(1) SECONDARY OR DESCENDING MESENTERIC VENOUS THROMBOSIS.

One of the first effects of partial or complete occlusion of the portal vein is necessarily a retardation of the circulation in the tributaries. This appears to be one of the chief factors in the secondary involvement of the mesenteric veins; at the same time, it cannot be assumed with safety that retardation of the blood-stream is the only factor, as there are numerous cases in which the mesenteric veins either did not become involved at all, or else only after a long interval.

When however a condition of sepsis is superadded to retardation in the mesenteric venous circulation, circumstances are much more favourable to the peripheral spread of the thrombosis from the portal vein.

If therefore it is assumed that portal thrombosis can result from obstruction alone (cirrhosis, perihepatitis, etc.) the process is much more likely to remain localised in such a case than if it is associated with sepsis: for in the former case when once a collateral circulation has been established the only stimulus for thrombosis has been removed, whereas in the latter, septic organisms continue to produce phlebitis and further thrombosis.

(2) PRIMARY MESENTERIC VENOUS THROMBOSIS.

Descending mesenteric thrombosis, as we have seen, is associated in most instances with a definite retardation of the blood-stream following portal obstruction, combined with a greater or less degree of sepsis.

In primary mesenteric thrombosis, on the other hand, we are dealing with a process in which at the onset the obstructive element is absent. With this reservation it may be said that there is no aetiological peculiarity of secondary mesenteric thrombosis that is not shared by primary.

Primary thrombosis generally arises from a phlebitis resulting from an infective process in the intestinal tract or the female pelvic organs, but there is a large number of cases for which no cause can be ascribed. The term 'marasmic thrombosis' has been

applied to some of these cases, but it is unsatisfactory in that it conveys no precise meaning.

If an analysis is made of the intestinal causes of venous thrombosis, it is found that the appendix is the most frequent source of infection: 26 cases (excluding two doubtful ones) attributable to this lesion have been collected in the present series. They may be tabulated as follows:—

The appendix was perforated	in 8 cases ¹
„ „ „ gangrenous	„ 8 „ ²
„ „ „ acutely inflamed	„ 3 „ ³
„ „ „ inflamed	„ 1 case ⁴
„ „ shewed old changes	„ 4 cases ⁵
Condition of appendix not stated (appendectomy <i>à froid</i>)	„ 2 „ ⁶
Abscess was certainly present in 12 cases out of 26.	

From the above table it is clear that the appendix was seriously affected in no less than 20 cases, or 77 % of the total; of the remaining six, four shewed signs of previous changes of an inflammatory nature. The figures seem to shew therefore that there is some relationship between the severity of the appendicular condition and the onset of a mesenteric phlebitis or thrombosis.

Gerster is of the opinion that certain cases that end fatally after operation for appendicitis are not fatal as a result of shock, but on account of venous septic thrombosis.

Mesenteric venous thrombosis complicating appendicitis may develop in several different ways. (1) The appendicular vein, or a vein in the wall of an appendix abscess, may become thrombosed, and the process spread by direct continuity (*a*) through the right colic to the superior mesenteric trunk, or (*b*) through the superior

¹ Polya (3 cases), Gerster, Deckart, original case No. 3, Jackson, Porter and Quinby's series (case No. 17), London Hospital P. M.'s 1910, No. 458.

² Polya, Gerster (2 cases), Hubschmann, Jackson, Porter and Quinby's series (case No. 13), London Hospital P. M.'s 1910, Nos. 931 and 997, and Sprengel.

³ Picot, original case No. 5, London Hospital P. M.'s 1909, No. 986.

⁴ Jackson, Porter and Quinby's series, case No. 16, and ? No. 26.

⁵ Hubschmann, Falkenburg, Jackson, Porter and Quinby's series (case No. 27), London Hospital P. M.'s 1911, No. 147.

⁶ Tschudy, Blake.

mesenteric from its origin: in either case the jejunal or ileal veins or both sets may become thrombosed in a retrograde manner from the main trunk.

(2) A septic embolus may be carried from a thrombosed appendicular vein through the right colic, across the main trunk of the superior mesenteric into one of the jejunal veins which opens nearly opposite the right colic¹; in this way the jejunal veins may be the first after the appendicular to be thrombosed.

(3) Septic emboli or bacteria may be carried directly to the portal vein, and initiate a descending thrombosis.

It is well recognised that after an appendix has been the seat of acute inflammation it rarely recovers completely, but usually remains infected, although the patient appears to be perfectly well. It is conceivable therefore that just as an appendix in such a state of quiescent infection may at any moment become acutely inflamed, so also at any moment it may suddenly cause an infective phlebitis in its wall, and a consequent spreading thrombosis.

The proximity of a vein to the wall of the abscess-cavity has undoubtedly an important bearing upon the development of thrombosis in certain cases. In case No. 3 (original) described above, thrombosis began in a small vein that arose from the lining of the abscess-cavity, while in one of the cases reported by Polya there was direct continuity between the abscess-cavity and the lumen of a vein.

A septic thrombosis originating in the appendix, like similar affections elsewhere, may remain localised. This point is well illustrated by case No. 5 (original): here the process was limited to the veins draining the lower end of the ileum.

In many cases of limited thrombosis complicating appendicitis it is probable that the symptoms produced are ascribed either to the primary lesion or to localised peritonitis. In this way slight cases are overlooked as they do not lead to infarction, but merely

¹ We are indebted to Polya for the above observation. His own words may be quoted:—‘Auch müssen wir als besonders bemerkenswert hervorheben, dass in dreien von unseren vier Fällen die retrograde Embolie die obersten Jejunalgefäße betraf, was sich durch die anatomischen Verhältnisse, nämlich die grosse Nahe der Einmündungsstellen der Vena colica d. und Venae jejunaes und durch den steilen Verlauf der zum obersten Jejunum ziehenden Venen erklären lässt.’

to oedema, hyperaemia and a few capillary haemorrhages in the affected segment of intestine¹.

A few cases are on record of mesenteric venous thrombosis following operation for hernia, free or strangulated. Zesas reviews seven cases, five after strangulated hernia², and two after operation for free hernia³, and observes that all of them concerned old subjects in whom affections of the intestine and atheromatous processes of the vessels came into question. A sixth case after strangulated hernia has been reported by von Baracz in a woman of 75. Ullmann reported a case probably of this nature, but the diagnosis was not confirmed as the patient recovered without further operation: mesenteric venous thrombosis was suspected from the occurrence of profuse bleeding from the rectum which began on the sixth day after operation for inguinal hernia, and lasted for 48 hours.

It is not difficult to explain the onset of venous thrombosis after a strangulation, as the two most important factors (stasis and sepsis) are present in the strangulated coil, and it is merely a question of the extension of the process to veins outside the hernia. It is not so easy to say however why this complication should follow an operation for free hernia. It is possible of course that during manipulation the parts were bruised, and the entry of organisms from the intestine facilitated in this way. The same suggestion is applicable in the cases of mesenteric venous thrombosis which followed laparotomy⁴ and gastro-enterostomy⁵.

One case is reported in which mesenteric venous thrombosis followed partial thyroidectomy⁵.

Affections of the female pelvic organs were responsible for mesenteric venous thrombosis in a number of instances, and though some, strictly speaking, belong to the secondary group (descending thrombosis), it will be convenient to discuss them all together under the present heading.

¹ A case probably of this nature is described in the London Hospital P. M. Records for 1911, No. 154.

² Cases reviewed by Zesas: Lauenstein, Pupovae, Hoffmann, Langer, Laroyenne.

³ Cases reviewed by Zesas: Quenu, Barthélemy.

⁴ Köster (reviewed by Kölbing).

⁵ Maylard.

The condition was reported after miscarriage	in 3 cases ¹
„ „ „ „ premature labour	„ 1 case ²
„ „ „ „ normal labour	„ 5 cases ³

If we compare these nine cases we are at once struck by the variability in the period of onset of intestinal symptoms after confinement: thus in the three cases of miscarriage the latent periods were 7 days, 6 weeks, and 6½ weeks; in the case of premature labour ‘several weeks,’ and in the five cases of normal labour, 23, 35, and 4 days, 4½ weeks, and ‘soon after.’ The question of the latent period will be considered at a later stage (*vide* ‘Symptomatology’).

Among the exceptional causes to which mesenteric venous thrombosis has been ascribed may be mentioned the following: non-ulcerating carcinoma of the rectum⁴, prostatic abscess of gonorrheal origin⁵, suppurative parotitis⁶, cellulitis of the neck⁷, uraemic ulceration of the intestine⁸, and typhoid fever⁹. It is remarkable that the last named disease, which on *a priori* grounds might be expected to play an important part in the aetiology of mesenteric thrombosis, has been reported in two cases only. Ulceration of the intestine that is so frequently present in typhoid fever, and phlebitis which not uncommonly follows the disease, appear to exercise but little influence upon the mesenteric veins.

Ulceration of the intestine from any cause very rarely leads to thrombosis. One case¹⁰ is on record in which stercoral ulceration of the sigmoid and rectum was associated with thrombosis of the inferior mesenteric and portal veins. In the same case there was found also a black sloughing appendix, but judging from the distribution of the thrombus it seems more likely that the origin of the process lay in the ulcers rather than in the appendix. It has been possible to find only one case¹¹ of venous thrombosis resulting

¹ Maygrier and Letulle, Lerat and Cleret, Garmsen.

² Mouchet.

³ Kölbing, Hilton Fagge, Broussin, Johst, and Jackson, Porter and Quinby's series (case No. 11).

⁴ Auriol (reviewed by Zesas).

⁵ Sprengel.

⁶ Goodheart.

⁷ McWeeney.

⁸ Castaigne (reviewed by Maclaure and Jacoulet).

⁹ Monro and Workman, Köster (latter reviewed by Kölbing).

¹⁰ London Hospital P. M.'s 1910, No. 931.

¹¹ Lagane's case No. 4.

from tubercular ulceration of the intestine. Lagane considers that in this case ulceration led to invasion of the venules of the intestinal wall by organisms, and that phlebitis was produced leading to thrombosis in the veins.

Abscesses in the pelvic meso-colon associated with false diverticula were held responsible in one case¹ for septic thrombosis of the inferior mesenteric vein: the clot extended as far as the termination of the splenic vein, but did not involve the superior mesenteric.

RETROGRADE VENOUS EMBOLISM.

A remarkable case of retrograde embolism of the mesenteric venous radicles has been reported by Woloschin. The mesenteric veins and their main tributaries were found to be free of clot, but thrombi containing liver cells were discovered in the capillaries of the subserous and submucous coats of a small coil of small intestine. The corresponding arteries and their branches were free. Woloschin believes that the liver cells reached the intestine as a result of injury to the liver, and venous regurgitation. This explanation is not altogether easy to accept, in that it implies the development of a pressure in the portal vein considerably above that in the mesenteric arterioles, a pressure sufficient to force the blood from the portal vein back through the capillaries. On the other hand, in the absence of a patent foramen ovale or signs of pulmonary embolism, one is at a loss to account for the presence of liver cells in the mesenteric capillaries. Woloschin's view derives a certain amount of support from a case reported by Gobiet. This author attributed thrombosis of the superior mesenteric vein in his case to retrograde venous embolism from the veins of the omentum which had been divided and ligatured in an operation for gastric ulcer. He says that the intra-thoracic pressure can be changed by coughing from a negative to a positive one, and the positive pressure thus produced be transmitted to the portal system. An embolus may in this way be carried back into a tributary vein.

¹ De Jong's case No. 8.

RESULTS OF VENOUS THROMBOSIS.

Out of 137 cases of mesenteric venous thrombosis infarction was found in 91. The 91 cases of infarction may be tabulated as follows:—

Trunk alone involved in 14 cases ('primary' thrombosis in 1 case, 'secondary' thrombosis in 13).

Tributaries alone involved in 21 cases (all 'primary').

Trunk and tributaries involved together in 56 cases ('primary' thrombosis in 9 cases, 'secondary' in 47 cases).

In other words, thrombosis in the mesenteric system resulted from portal thrombosis or morbid conditions in the portal veins in 60 cases, and occurred by itself in 31 only.

In many cases where the portal vein has been thrombosed a comparatively small sector only of the mesentery and intestine has been infarcted, the veins draining that part alone being thrombosed in direct continuity with the superior mesenteric and portal trunks, while the rest of the mesenteric radicles from other parts remained free.

In other cases again the trunk and the greater part of the mesenteric tributaries were thrombosed but only a small number of the venous radicles. In such cases the infarct was limited to this small region of occluded radicles, while the rest of the intestine was simply hyperaemic and oedematous.

It may happen¹ in the event of coexistent thrombosis of the portal, superior, and inferior mesenteric veins that the small intestine is infarcted almost in its entirety, but that the large intestine shews lesions of a minor degree (e.g. oedema, engorgement, etc.).

Though infarction of a large segment of the small intestine sometimes follows closure of the trunk only of the superior mesenteric, yet it is much more usual for the infarcted area to be limited to a relatively small segment till the process has spread to the tributaries.

The 46 cases of venous thrombosis in which the intestinal lesion did not advance to the stage of infarction are tabulated below:—

¹ De Jong's case No. 8.

Trunk alone involved in 13 cases ('primary' thrombosis in 2 cases, 'secondary' in 11).

Tributaries alone involved in 13 cases ('primary' thrombosis in 11 cases, and 'secondary' retrograde embolism in 2).

Trunk and tributaries involved together in 20 cases ('primary' thrombosis in 14 cases, and 'secondary' in 6).

Thus the 46 cases consisted of 27 in which the process arose in the mesenteric system, and 19 in which thrombosis was initiated in the portal vein.

On comparing the two tables therefore we see that there is a smaller percentage of 'secondary' thromboses that lead to minor lesions than to gross lesions in the intestine, as would be expected from the limitation in the collateral channels in 'secondary' thromboses as compared with 'primary' ones.

The lesions in the intestine in these milder cases consisted of oedema and engorgement—often intense,—ulcerations, submucous haemorrhages, or changes of a milder type still. In some there were no intestinal lesions at all, but these for the most part concerned cases where only a few small tributaries were occluded.

The distribution of the intestinal changes will be dealt with in the section upon 'Morbid Anatomy.'

(b) ARTERIAL THROMBOSIS AND EMBOLISM.

Serious damage in the intestine takes place in the large majority of cases after closure of any but a small branch of the superior mesenteric artery. Out of 197 cases collected in this series, 106 shewed infarction, 36 haemorrhages or extreme engorgement, and 10 ulceration of the mucosa: actual perforation of the intestine was found in five cases only.

Out of the whole number of cases perforation was associated with infarction in two¹, ulceration with infarction in one², and ulceration with haemorrhages in two³. Apart from these five cases the above figures are mutually exclusive, so that out of a total of 197 cases, 157 shewed more or less gross intestinal changes. The remaining 40 comprise those cases which were

¹ Two cases reported by Adénot.

² Kussmaul.

³ Parensky, Schnitzler.

diagnosed but did not prove fatal, those in which no actual clots were discovered in the arteries at autopsy though perhaps present in small branches, and finally those few remarkable cases in which a complete collateral circulation had been established.

The explanation for the high percentage of serious effects resulting from mesenteric arterial obstruction is to be found in the discovery made independently by Litten and Faber¹ in 1875. The experimental work of these authors will be referred to at a later stage: suffice it to say here that the superior mesenteric artery was shewn by them to be an 'end-artery' functionally though not anatomically. For this reason it is very exceptional to find that a complete collateral circulation has been established after the closure of any but a small branch.

Ten cases are on record in which a collateral circulation had been established before death, but in six of these it ultimately proved insufficient.

In Virchow's first case the superior mesenteric artery was found at autopsy to have been transformed into a fibrous cord: no changes were found in the intestine.

In his second case no essential changes were found in the intestine in spite of the fact that practically the whole of the artery was full of firm clot to within half an inch of the intestine. This case is further remarkable in that it is the only certain one on record in which the source of the embolus was the pulmonary vein.

In Chiene's case the coeliac axis, superior and inferior mesenteric arteries were all obliterated at their origins by very old thrombi. There was a pear-shaped aneurism extending from the origin of the left renal artery to the bifurcation of the aorta. A collateral circulation had been established through the haemorrhoidals, the middle and left colics, and splenic artery, and by the last dorsal on the right side with the ileo-colic artery. The superior haemorrhoidal equalled in size the femoral at Poupart's ligament. No intestinal changes were present.

¹ The following quotation may be given from Faber's work:—'*Die art. mes. sup. verhält sich mithin wegen der im Verhältniss zu ihrer weiten Ausbreitung geringfügigen Anastomosen bei ihrer Verstopfung wie eine wirkliche Endarterie.*'

In case No. 6 (original) the coeliac axis and superior mesenteric were completely occluded by old, firm, organised thrombi. The inferior mesenteric was narrowed at its origin, but patent. All the three vessels took origin from the wall of an aneurism of the aorta. Unfortunately the collateral channels were not dissected out. There were no changes in the stomach or intestines.

In Karcher's case the patient died two months after complete embolism of the superior mesenteric artery, apparently as a result of exhaustion following sepsis in an amputation stump.

Cohn's case shewed a firm fibrous clot in a branch of the superior mesenteric artery. The patient died from infarction.

The history in Schnitzler's case extended over a period of four or five years. An exploratory laparotomy was performed at the onset of the acute symptoms, and the patient survived for six weeks. An old, firm clot was found at the autopsy in the trunk of the superior mesenteric artery, and the inferior mesenteric was narrowed. Infarction of the intestine had supervened. The inferior pancreatico-duodenal and inferior haemorrhoidal arteries had provided the collateral blood-supply.

Kaufmann's patient, a woman of 80, died from infarction of the whole of the small intestine and a large part of the colon, resulting from failure of compensation in the blood-supply after an old embolism of the superior mesenteric trunk.

Howse's patient had complained of abdominal pains for months before death, with diarrhoea and melaena at times: there had been vomiting (the vomited matter being free from blood) for the last three weeks of life. At the autopsy a firm thrombus was found in the trunk of the superior mesenteric artery, extending into the aorta. There was ulceration of the ileum and ascending colon.

In Lavarán's case, a man of 41, an old, firm clot was found in the superior mesenteric artery: the middle 50 cms. only of the small intestine shewed infarction.

In Kölbing's case, in spite of closure of the superior mesenteric trunk by an embolus, the intestine shewed only slight reddening of the follicles and slight oedema. There must have been therefore a certain degree of compensation.

Although it is probably true that the superior mesenteric

artery and its main branches behave as 'end-arteries' in the vast majority of cases, yet as will be seen later, there is evidence both experimental and clinical for the belief that infarction of the intestine does not necessarily follow closure of small branches. Oedema, haemorrhages into the intestinal wall, ulceration of the mucosa, greater or less extravasation of blood into the lumen, and other minor changes not uncommonly follow occlusion of small branches, and lead to grave clinical manifestations: these however are not always fatal. One case¹ is on record in which stenosis of the jejunum was attributed to an old infarction. Although recovery is known on experimental evidence to be possible after engorgement and extreme oedema of the intestine, yet it is doubtful whether recovery from actual infarction ever occurs. Deckart² says that an infarct, however narrow, acts as a mechanical obstruction, but Gobiet³ is of the opinion that the obstruction offered by a narrow infarct may be overcome. It is just possible that a very small infarct may be followed by fibrosis and recovery, and in such a case the fibrosis would lead to stenosis of the intestine and consequent symptoms of incomplete obstruction.

The mesenteric arteries may be occluded in a variety of different ways, the most important being

- (1) by thrombosis, and
- (2) by embolism.

Less frequent causes of obstruction are extreme endarteritis⁴, arterio-sclerosis⁵, atheroma⁶ in the arteries themselves or in the aorta immediately around their points of origin. These rarer conditions sometimes lead secondarily to thrombosis, and will on this account be considered under that heading.

In view of the frequency with which thrombosis follows embolism, and the intimate way in which the thrombus may become grafted on to an embolus, it is often impossible to say with certainty whether a case was primarily one of embolism or of thrombosis; moreover, it sometimes happens that embolism of small branches follows obstruction (whether by embolus or thrombus) of the parent trunk⁷.

¹ Langerhans.

² Deckart, *loc. cit.* S. 558.

³ Gobiet, *loc. cit.* S. 1560.

⁴ Leclerc and Cotte. ⁵ Jackson, Porter and Quinby's series, No. 3 and ? No. 9.

⁶ Lagane's series, Nos. 2, 5, 6, and 9.

⁷ Altmann.

(1) THROMBOSIS OF THE MESENTERIC ARTERIES.

The aetiology of mesenteric arterial thrombosis is often a matter of great difficulty, and in certain cases it is possible only to suggest the relationship between the process and coexistent morbid-anatomical conditions.

The most common cause of thrombosis in the mesenteric arteries is embolism, the vessel and its branches becoming filled by the peripheral spread of the clot in direct continuity with the embolus. (The subject of embolism will be considered later.)

Atheroma of the aorta may occasion thrombosis (*a*) through the narrowing or even complete obliteration of the origin of the vessel as the result of the formation of a calcareous plate¹ in this region: or (*b*) by the direct spread of a thrombus into the mesenteric artery from an atheromatous 'ulcer' in the aorta².

Aneurism of the aorta has been responsible in several instances for obstruction of the mesenteric arteries. This obstruction has been brought about either by the spread of the fibrinous lining of the aneurism into the mouth of the vessel³, or more frequently by the gradual constriction of the origin of the arteries as they pass through the wall of the sac⁴. In the latter event thrombosis may be very limited, the peripheral part of the trunk and the branches remaining free. Thrombosis in these cases is probably due to inflammatory changes such as are known to occur about the walls of an aneurism.

Atheroma of the mesenteric arteries themselves has been reported in a few cases⁵ of thrombosis, but it is generally regarded as rare⁶. In one case the origin of the middle colic was obliterated by a calcareous plate: in a second the thrombus rested

¹ Richards.

² Cases Nos. 4 and 7 (original), Councilman, Wood, and Jürgens.

³ Osler.

⁴ Case No. 6 (original), Jackson, Porter and Quinby's series, No. 2, Virchow, Chiene, and possibly Halipré.

⁵ Adénot, Firket and Malvoz, Labbé, and Trübel.

⁶ Lagane, however, disagrees with the accepted view regarding the frequency of mesenteric atheroma, and has published no less than four original cases (Nos. 2, 5, 6, and 9) in which either the trunk or branches were the seat of atheromatous processes. It is noteworthy in this connection that infarction may be caused by atheroma or advanced arterio-sclerosis, apart from thrombosis.

upon an atheromatous base at one of the bifurcations of the superior mesenteric: while in a third there were atheromatous plaques in the artery.

Endarteritis, if not an exciting cause¹, certainly exercises a predisposing influence upon the production of thrombosis². It has been named as the cause in three or four cases³, and was probably responsible for the process in a few others⁴. The diminution of the blood-supply associated with the narrowing of the lumen and the roughening of the intima no doubt has some effect upon the blood-stream, increasing the chances of coagulation. The coexistence of myocarditis accentuates this tendency by further enfeebling the circulation.

The view is gaining ground abroad⁵ that arterio-sclerosis is of great importance in the aetiology of mesenteric arterial thrombosis, and it is held responsible by several authorities⁶ for a prodromal clinical condition which has received the expressive, though somewhat unwieldy, name of '*Dyspragia intermittens arteriosclerotica intestinalis*'.

¹ Obliterative endarteritis in the mesentery is considered to be a rare condition by Rokitsansky and Huchard (quoted by Schnitzler).

² To judge from the results of Lagane's work, this applies with particular force to the branches in the intestinal walls. Case No. 1 in Jackson, Porter and Quinby's series is a good example of thrombosis caused by endarteritis.

³ Nos. 14 and 18 of Jackson, Porter and Quinby's series, Case No. 1 (original), and Meyer.

⁴ Beckmann, Faber (a woman of 82), and Gordon.

⁵ Orthner, Zesas, Sauvé and others.

⁶ Authors just mentioned, and Schnitzler, Breuer (quoted by Zesas), and Lagane.

⁷ Orthner made a very thorough investigation into the subject of arterio-sclerosis and endarteritis as affecting different parts of the body, and drew a parallel between the clinical manifestations attributed to them. Thus, he believes that certain cases of Stokes-Adams' disease ('*Dyspragia intermittens arterio-sclerotica medullaris*'), intermittent claudication ('*Dys. int. art. cruralis*'), intermittent amaurosis ('*Dys. int. art. retinae*'), angina pectoris ('*Dys. int. art. cordis*'), and the disease he has named '*Dys. int. art. intestinalis*' are all manifestations of the same disorder in different organs, and that they depend upon two main factors:—sclerosis of the vessels of the part concerned, and reflex vaso-constriction. He adds, however, that the symptoms may be produced by other conditions than sclerosis of the arterioles, viz. by partial or complete closure of the main artery of an organ by embolus or thrombus with permeability of its main branches. In view of the importance he attaches to the reflex nervous element it is an interesting speculation as to whether Reynaud's disease may not be brought into line with the other affections mentioned.

Before we can accept arterio-sclerosis as a common cause of thrombosis in the mesenteric arteries it is necessary to ascertain in the first place the frequency with which these vessels are affected by sclerosis, and secondly the effect of arterio-sclerosis (as distinguished from endarteritis and atheroma) upon the blood-stream.

Orthner quotes a number of authorities in his discussion upon the frequency of mesenteric arterio-sclerosis, and their opinions appear to differ very considerably. Thus Hasenfeld considers that arterio-sclerosis is frequent in the splanchnic vessels 'in small and medium sized masses' only on microscopic examination, but that considerable sclerosing processes are much rarer than in the aorta, in the arteries of the extremities, and of the brain¹: further, that arterio-sclerosis may be present in the splanchnic vessels and absent from the radials and temporals². Lohnstein, Bregmann, and Mehnert on the other hand do not lay any special stress upon the mesenteric arteries as a seat of sclerosing processes. This difference of opinion can no doubt be partly explained on the supposition that the term 'arterio-sclerosis' is used by some authors as a synonym for 'atheroma,' while it is applied by others to a general sclerosis of the wall of the vessel. Lagane, who made a most exhaustive study of the diseases of the mesenteric vessels, demonstrated the existence of a series of pathological changes in the mesenteric arteries and their branches ranging from a slight degree of thickening of the intima (endarteritis) on the one hand, to gross atheroma on the other, arterio-sclerosis with thickening and loss of elasticity of the intima, media and adventitia occupying a somewhat intermediate position. By examining sections of the wall of the intestine he was able to find definite pathological changes in the walls of the arterioles (with or without thrombosis of these vessels) in cases that shewed no gross lesions of the nature of embolism or thrombosis in the

¹ Hasenfeld (quoted by Orthner) says :—' Die Arteriosklerose der Splanchnicus-gefäße in geringem und mittlerem, nur bei mikroskopischer Untersuchung wahrnehmbarem Masse recht häufig ist. Starke sklerotische Veränderungen sind dagegen viel seltener als an der Aorta, den Arterien der Extremitäten und des Gehirns.'

² The patchiness in the distribution of arterio-sclerosis in different parts of the body is emphasised by Jackson, Porter and Quinby, Lagane, and others.

mesentery. His researches suggest that if systematic histological examination had been made in many of the cases where no thrombus or embolus was found, but which had manifested signs and symptoms suggestive of a vascular obstruction, definite thickening and even obliteration of the smaller arterial branches by extreme endarteritis might have been discovered. The main causes given by him for sclerosing changes in the arteries and their branches are syphilis (in the young) and old age. Typhoid fever was held responsible for chronic arteritis in one of his cases.

To sum up therefore it may be assumed that atheroma (gross patches beneath the intima, and fatty and calcareous plates giving rise to local, asymmetrical diminution of the lumen of the vessel) is much rarer than in the cerebral arteries, but that sclerosis of the coats,—particularly the adventitia and intima,—producing loss of elasticity in the wall, is not uncommon in the small vessels of the mesentery and intestinal walls.

One of the most important effects of arterio-sclerosis upon the affected vessel is to deprive it of its power of dilating in response to reflex stimuli. Thrombosis probably supervenes as a result of the chronic inflammatory changes in the wall of the vessel, changes analogous to those found in the wall of an aneurism.

Sauvé expresses the opinion that visceral arterio-sclerosis is the most common cause of mesenteric arterial thrombosis, but that complete thrombosis from sclerosis is preceded by a long period of rigidity without obstruction; in this view he is in agreement with Lagane.

It is during the long preliminary period of sclerosis that symptoms arise which constitute the clinical entity referred to above.

With regard to ‘*Dyspragia intermittens arteriosclerotica*¹ *intestinalis*’ Rosengart is quoted by Zesas as follows:—‘Its morbid-anatomical equivalent is a local anaemia of the larger or smaller vessels of the intestine at a time when greater performance of work is required of the intestine.’ That is to say, the branches of the mesenteric arteries that have become narrowed by sclerosis are capable of carrying sufficient blood to the intestine for ordinary purposes, but are incapable of dilating in an emergency.

¹ Or ‘*angiosclerotica*.’

The result is that when there is an increased demand for blood the relative anaemia produces spasmodic pains with motor insufficiency in the intestine; this is followed by reflex vasoconstriction in the arterioles and capillaries, causing further anaemia. This condition appears to have obtained as a result of extreme mesenteric or parietal endarteritis in the cases reported by Leclerc and Cotte, Merkel, and Lagane (Nos. 2 and 3 of his series). The clinical aspect of mesenteric arterio-sclerosis will be referred to later.

Mesenteric arterial thrombosis was ascribed in one case¹ to the pressure of an enlarged gland upon the vessel.

RESULTS OF MESENTERIC ARTERIAL THROMBOSIS.

The morbid anatomy of mesenteric vascular occlusion will be considered at a later stage, but it will be convenient at this point just to mention the kind of effect produced.

Considering the frequency with which thrombosis is caused by, or at all events associated with embolism, it is not surprising, for reasons that will be seen later, that the superior mesenteric artery is thrombosed more often than the inferior.

(a) *Thrombosis of the Superior Mesenteric Artery alone.*

Out of this group of 61 cases (including 22 cases that may have been due primarily to embolism) haemorrhagic infarction was found in 34 (including three² with perforation of the ileum), haemorrhages or intense engorgement in 12, mucous catarrh in one, and no changes in the intestine in three³. In three cases⁴ the patient recovered, in five no autopsy was held, and finally in three the condition of the intestine was not mentioned.

Of the 34 cases that shewed haemorrhagic infarction the trunk of the artery was closed in 21, one or more main branches in 10, and small branches in three: in one of these cases although only the smaller branches were thrombosed, the larger ones were partly obliterated by sclerosis⁵.

¹ Lépine.

² Blake, No. 18 in Jackson, Porter and Quinby's series, London Hosp. P. M.'s 1908, No. 1230.

³ Case No. 6 (original), and Virchow (2 cases).

⁴ Gordon, Gosset, and Neumann.

⁵ No. 3 in Jackson, Porter and Quinby's series.

In all the cases in which the thrombus did not affect the main trunk, the branch or branches occluded corresponded with the infarcted segment.

As regards the 12 cases in which no infarction was found, the trunk was absolutely closed in eight¹, and partially closed in one²: the corresponding branches were thrombosed in two³, and in the last⁴ no thrombus was found to account for the engorgement, as no autopsy was permitted. There is very little doubt that had these 12 patients lived even a short time longer, actual infarction would have supervened, the process having only just stopped short of this condition.

The three cases⁵ in which no changes were found in the intestine have already been discussed above.

From the above figures it is obvious that thrombosis of the superior mesenteric artery or one or more of its main branches leads in the vast majority of cases to grave consequences in the intestine.

(b) *Thrombosis of the Superior and Inferior Mesenteric Arteries together.*

This is a rare condition, only six cases having been recorded. In the first (described above)⁶ there were no intestinal changes in spite of complete obliteration of the origins of both arteries. In the second⁷, due like the first to aortic aneurism, any effects upon the intestine that may have been produced are not reported by the reviewer⁸, though we are told there had been signs of intestinal obstruction. In the third case⁹ there were adherent thrombi in the trunks of both arteries, and gangrene with perforation of the ileum; the walls of the intestine were infiltrated with blood, and there was no sharp line of demarcation; the duodenum and jejunum were distended with gas. In the fourth case¹⁰ branches of the superior mesenteric and the trunk of the inferior were occluded by firm, red thrombus, resulting in gangrene of 10½ feet of the lower jejunum and upper ileum with perforations of the

¹ Councilman (2 cases), No. 2 in Jackson, Porter and Quinby's series, Mader, Mollard and Monod, Pieper, Triebel, and Virchow.

² Altmann.

³ Goodheart and Lépine.

⁴ van Hook.

⁵ Virchow (2 cases), and case No. 6 (original).

⁶ Chiene.

⁷ Dinkler.

⁸ Talke.

⁹ Fischer.

¹⁰ Kiliani.

latter. In the fifth case¹ there was organised clot in the origins of both arteries; compensation had taken place in the vascular supply, but had finally proved insufficient; the whole of the jejunum and ileum and ascending colon were infiltrated with blood, and ulcerated. In the last case² there was very advanced atheroma of the abdominal aorta, and the superior and inferior mesenteric arteries were described as being occluded by thrombi of senile endarteritis. The intestinal mucosa was enormously hyperaemic from stomach to anus, and the intestines were full of blood; there was no trace of gangrene.

(c) *Thrombosis of the Inferior Mesenteric Artery alone.*

It has been possible to collect only five cases of this nature. In the first³, the middle and left colic branches were completely occluded, and gangrene with large perforations of the wall followed in the corresponding portion of the colon; the lower two-thirds of the colon was described as being shrunken. The second case⁴ shewed an annular area of gangrene in the lower part of the descending colon, with a large perforation; the arteries of supply were thrombosed. In the third case⁵ there were also gangrene and perforation of the descending colon, resulting from thrombosis of the inferior mesenteric trunk. In the fourth⁶, the mucosa of the descending colon, sigmoid and rectum was loosened, dark red, and contained numerous haemorrhages. The causal lesion was an adherent clot in the origin of the inferior mesenteric. The last case⁷ shewed a rather peculiar distribution of the gangrene, only in part corresponding with the vessels that were found thrombosed. The caecum and to a less extent the hepatic and splenic flexures were infarcted and necrotic, while the sigmoid and rectum were free. The inferior mesenteric was thrombosed.

A striking fact about this last group is the large percentage of perforations that followed vascular occlusion: they were found in no less than three out of the five cases, i.e. in 60 %. The explanation probably lies in mechanical influences, the colon being much more subject to tension than the small intestine, owing to the accumulation of faeces.

¹ Schnitzler.

² Merkel.

³ Adénot.

⁴ *ibid.*

⁵ Elliot.

⁶ Hegar.

⁷ Pinner.

(2) EMBOLISM OF THE MESENTERIC ARTERIES.

Embolism of the mesenteric arteries and their branches is a much commoner condition than thrombosis. Out of 187 cases of mesenteric arterial occlusions embolism was responsible for 126, exclusive of those cases mentioned above that may have been originally embolic.

When we compare the relative frequency of embolism as affecting the superior and inferior mesenteric arteries the disparity is at once evident, the superior artery or its branches being involved in 119 cases, the inferior alone in two only, and the two vessels together in five.

Gallavardin gave the following explanations for this disproportion in the incidence of embolism in the two vessels:—

(1) The superior mesenteric arises from the aorta above the inferior, and therefore has an earlier opportunity of intercepting an embolus.

(2) The superior mesenteric measures 9 mm. in diameter, whereas the inferior measures only 3·5 to 5 mm.

(3) The superior artery runs a course nearly parallel to that of the aorta, while the inferior leaves that vessel at an angle of about 45 degrees.

A consideration of the above explanations leads one to attach less weight to the first than to the second and third, for if the first were really of great moment one would expect to find the incidence of embolism in general diminish as one passed from the upper to the lower parts of the body. The relative sizes of the two mesenteric arteries and their different courses however do appear to be factors of great importance in determining the difference in the incidence of embolism.

SOURCE OF THE EMBOLUS.

The embolus may be liberated from one of three sources:—

(1) The lungs or pulmonary vein.

(2) The heart: in the vast proportion of cases the left side, but possibly in very exceptional cases the right side through a patent foramen ovale.

(3) The aorta.

The first is an extremely rare point of origin, only one undoubted instance¹ being on record: yet there are two others which lend themselves to the same interpretation in view of the course of the symptoms. In the first², signs of pulmonary embolism followed the act of defaecation in a man suffering from prolapsed piles. Shortly afterwards, signs suggestive of mesenteric embolism (diarrhoea, melaena, resistance and tenderness in the right iliac fossa) supervened. The diagnosis was not confirmed as the patient recovered. The second case (original No. 2) has already been described.

The second possible origin of an embolus is by far the commonest. Acute and chronic endocarditis, affecting the mitral and aortic valves, are responsible for the majority of cases.

Talke in his discussion upon the frequency of mesenteric embolism caused by endocarditis quotes two series of autopsies reported by Sperling and Ginsburg respectively. Sperling out of 300 cases of endocarditis found 84 with emboli in some organ or another, including five with embolism in 'liver and intestine.' Ginsburg out of 250 cases of endocarditis found embolism in 85, the superior mesenteric artery being involved in three. It is unfortunate for our purpose that the liver and intestine should have been grouped together in the first series: but supposing we assume that two of the five cases concerned the intestine,—not an excessive number considering there were three in the other (smaller) series,—that will give a total of five cases of mesenteric embolism in 550 of fatal endocarditis, or about .9 %.

This at first sight seems to be a rather high percentage, but it probably includes not only cases in which the trunk was occluded, but also those in which one or more of the main branches only were occluded.

Acute or chronic endocarditis was responsible probably for 70 of the cases of mesenteric embolism in the present series. In the majority of the cases the cardiac lesion was or had been associated with rheumatic fever.

Pyæmia and malignant endocarditis, strange to say, are among the rarer causes of mesenteric embolism. Out of the present

¹ Virchow (case already referred to above).

² Bayer.

series only 11 cases¹ can be ascribed to these causes. Gallavardin ascribes the apparent rarity of malignant endocarditis as a causal agent in mesenteric embolism to the fact that such cases are reported under the title of 'Aneurism of the mesenteric artery': septic emboli lead to dilatation in the vessel wall as a result of a localised acute arteritis. In one case² of malignant endocarditis there was an aneurism of the superior mesenteric probably associated with embolism of the inferior.

Endocarditis was associated with clots in the left side of the heart in eight cases³ and it is probable therefore that in some the emboli consisted not of fragments of the mitral or aortic valves but of the clot. Evidence in support of this view is afforded by the occurrence of 19 cases in which clots were found in the left side of the heart, apart from valvular disease.

The third possible source of emboli is the aorta. Atheroma of this vessel was to blame in most of the remainder of the cases, either through the separation of a fragment of a plaque, or of a portion of thrombus attached to an atheromatous 'ulcer'⁴. In the same way portions of clot from the interior of an aortic aneurism⁵ have become impacted in the trunk or one of the branches of the superior mesenteric.

RESULTS OF EMBOLISM OF THE MESENTERIC ARTERIES.

Out of 60 cases shewing embolism of the trunk of the superior mesenteric haemorrhagic infarction followed in 45: of the remainder, severe haemorrhages in the walls of the intestine were reported in seven, haemorrhages with perforation in one, slight reddening of the mucosa in two, and in five the changes are unknown.

¹ Malignant endocarditis was found in the following:—London Hosp. P. M.'s 1907, No. 827, 1910, No. 123, and No. 500, 1911, No. 50. The pyaemic or probably pyaemic cases were:—Arnstein and Troschewsky (stab wound), Cramer (puerperal pyaemia), Faber (suppuration in amputation stump), Hegar (after myomectomy), No. 22 in Jackson, Porter and Quinby's series (suppuration about jaw), London Hosp. P. M.'s 1908, No. 1153 (pyaemia from tonsillitis), and case No. 2 (original).

² London Hosp. P. M.'s 1910, No. 500.

³ Gimard, Karcher, Lochte, London Hosp. P. M.'s 1909, No. 529, and 1910, No. 802, Merkel, Sillitti, and Virchow.

⁴ Councilman, Tschudy and Seitz, Osswald.

⁵ Sievers, and probably Packard, and Parensky.

One or more large branches were obstructed by emboli in 28 cases, leading to infarction in seventeen, haemorrhages in three, haemorrhages with ulceration in two, ulceration in one, and perforation in two; in three cases the condition of the intestine is not recorded.

35 cases have been collected which shewed either probable or certain embolism of small branches of the mesenteric arteries. Out of these, fifteen were diagnosed upon more or less typical signs and symptoms, but never confirmed, nine shewed haemorrhages into the mucosa, five ulceration, four infarction, one a perforation, and one simply swelling of the mucosa.

There are only two undoubted cases of embolism of the inferior mesenteric artery alone. In the first¹ the trunk was occluded, probably by a portion of clot from a thrombus in the left auricle. The last 2½ feet of the ileum, together with the whole of the large intestine down to the anus, was purple and infiltrated with blood. This case is of interest, shewing as it does that the lower part of the small intestine may derive part of its blood-supply from the inferior mesenteric. In the second case² there were atheromatous 'ulcers' of the aorta covered by fibrinous deposits. The closure of the inferior mesenteric had led to early gangrene of the corresponding intestine.

EMBOLISM OF BOTH MESENTERIC ARTERIES TOGETHER.

This is a very rare condition, two certain cases only being on record. In the first³ there was chronic endocarditis, with embolism of several large arteries besides the two mesenterics. Practically the whole of the intestine was infarcted. In the second⁴ both trunks were closed by emboli, and the whole intestine (large and small) was dark red and very distended.

In a third case⁵ there were infarcts in the mesentery and meso-colon, with corresponding engorgement and distension of the intestines, but the emboli could not be looked for as no autopsy was permitted. The second doubtful case⁶ was a patient with puerperal pyaemia. A septic polypoid thrombus was found

¹ Gerhardt.

² Tschudy and Seitz.

³ Bühler.

⁴ Westhoff.

⁵ Munro.

⁶ London Hosp. P. M.'s 1911, No. 50.

attached to the aortic valves, and there were infarcts in the spleen and kidneys: petechial haemorrhages were found throughout the intestines.

The consideration of mesenteric embolism would not be complete without some reference to certain of the more severe forms of purpura to which it appears to be allied. The relationship of the two conditions has been discussed at great length by Talke who contends that many cases of purpura are due to embolism.

Up to the present we have no certain knowledge as to the cause of Henoch's purpura, though various opinions have been put forward¹. As regards the embolic theory which immediately concerns us, it may be said that there is both experimental and clinical evidence in support of it. Thus a number of cases with purpuric manifestations have been reported in which emboli were definitely found in branches of the mesenteric arteries, or in which the presumption was very strong that embolism had occurred in them. Out of 14 such cases collected in the present series, mesenteric embolism was present for certain in eight², ulceration of the intestine was found in three³, and haemorrhages into the mucosa in one⁴: in another the clinical course was of a type strongly suggestive of embolism⁵, and finally in one case⁶ the diagnosis, unconfirmed by autopsy, has been called in question, resting as it did upon rather scanty evidence.

The source of the emboli, or supposed emboli, was in the majority of cases the heart. Endocarditis was held responsible in nine⁷, blood-clot in the heart in three⁸, and a polypoid thrombus in the aorta in one⁹: in one case¹⁰ no mention is made of the heart or aorta as a probable source of the emboli.

¹ Talke quotes the views of several authorities: thus it is believed by Sortais to be a toxæmia from the gastro-intestinal tract, or from bacterial infection: by Silbermann to be due to primary changes in the blood: while v. Kogerer and Riehl ascribe it to changes in the vessel-walls and Kraus to embolism (*loc. cit.* S. 756—763).

² Deckart, Edler, Gimard, No. 25 in Jackson, Porter and Quinby's series, Talke, Wittmann, London Hosp. P. M.'s 1908, No. 1153, 1910, No. 500.

³ Booth, Wagner, Wikner.

⁴ London Hosp. P. M.'s 1911, No. 50.

⁵ Moos.

⁶ Henderson.

⁷ Booth, Deckart, Edler, Henderson, London Hosp. P. M.'s 1908, No. 1153, 1910, No. 50, and 1911, No. 50, Moos, Wittmann.

⁸ Gimard, No. 25 in Jackson, Porter and Quinby's series, Wagner.

⁹ Talke.

¹⁰ Wikner.

Although it would be too sweeping an assertion to say that all severe cases of purpura were due to embolism, yet the above evidence combined with that derived from experiments—to be referred to later on—shews that some cases of purpura with intestinal manifestations are certainly embolic in origin.

(c) COEXISTENT OCCLUSION OF ARTERIES AND VEINS.

There are only about 22 cases on record in which both arteries and veins in the mesentery were occluded. We are indebted to Taylor for the report of the first case which was published in 1881. In 1892 Sprengel endeavoured to shew that haemorrhagic infarction resulted from a single (i.e. arterial or venous) vascular occlusion, but anaemic gangrene from a double (i.e. arterial and venous) one. He further tried to correlate the two conditions with separate clinical types—the first (haemorrhagic infarct) with symptoms and signs of intestinal haemorrhage (diarrhoea, vomiting, melaena, haematemesis), and the second (anaemic gangrene) with those of paralytic intestinal obstruction.

These varieties of the disease will be considered later; suffice it to say at this stage that the clinical types described by Sprengel do not correspond necessarily with the pathological conditions that were supposed to produce them.

Though at operation or autopsy both arteries and veins may be found to be occluded, it sometimes happens that the obstructions in the two classes of vessel are of different date. Thus in eight¹ of the cases venous thrombosis was believed after examination to be secondary to arterial thrombosis or embolism, while in two² the arterial obstruction was considered to be secondary to venous thrombosis. In one case³ the clots in both artery and vein were old, though the tributaries of the vein had become thrombosed as a terminal event.

¹ Jackson, Porter and Quinby, No. 1, Osswald, Saxer, Taylor, Meyer, Lagane, Nos. 3, 8 and 9.

² Fedorowitsch, Taravellier.

³ Sprengel.

The distribution of the vascular occlusions varied considerably. In some cases both trunks (arterial and venous) were occluded, while in others the trunk of the artery and tributaries of the vein, or the trunk of the vein and branches of the artery, were involved together. In some finally branches of the artery and tributaries of the vein were thrombosed. It would serve no useful purpose to express the various combinations of vascular lesions in tabular form, as the cases concerned are so few in number. So far as one may conclude from the 22 cases however, branches and tributaries are both occluded far more often than their respective trunks. Arterial occlusion in these cases was not uncommonly associated with definite sclerosing processes in the vessel walls.

RESULTS.

In 19 out of the 22 cases the double vascular occlusion led to more or less extensive haemorrhagic infarction of the small intestine. In seven¹ of the cases practically the whole of the small intestine was infarcted, while in others² the extent of the infarct varied from 30 cms. to a metre. In addition to the small intestine or part of it, the caecum was infarcted in one case³, and part of the ascending colon in another⁴.

In the three remaining cases⁵ anaemic gangrene was present. It was very limited in extent in all cases, the caecum, 10 cms. of the lower ileum, and 4 or 5 cms. only of the ileum being involved respectively. In this respect the intestinal lesions presented a very marked contrast with those where haemorrhagic infarction was present.

The morbid anatomy of haemorrhagic infarct and anaemic gangrene will be dealt with later (*vide* 'Morbid Anatomy').

¹ Grosskurth, Osswald, Saxer, G. M. Smith, Taylor, Fedorowitsch, Taravellier.

² Deckart, Jackson, Porter and Quinby, Nos. 1, 4, 14 and 17, Patal, Sprengel, Wakefield, Lagane, Nos. 1, 3, 8 and 9.

³ Fedorowitsch.

⁴ Jackson, Porter and Quinby, No. 4.

⁵ Niederstein, Sprengel, Meyer.

SECTION III

MORBID ANATOMY

ALTHOUGH we have already considered briefly some of the outstanding features of the lesions resulting from occlusion of the mesenteric vessels, yet it will be convenient at this stage to discuss rather more fully both the lesions themselves and the different conditions in the affected vessels.

For the sake of treatment the subject may be subdivided under the following headings:—

- (1) Changes in the vessels, the distribution of the clot and its relation to lesions in the mesentery and intestine.
- (2) Changes in the mesentery and glands.
- (3) Changes in the walls and contents of the intestine.
- (4) Peritoneal changes.
- (5) Bacteriology.

(1) VESSELS. (A) VEINS.

Thrombosed mesenteric veins are as a rule not merely filled but distended with blood-clot, and if rolled between the fingers give the impression of firm hard cords running through the mesentery. This cord-like consistence is not entirely due to blood-clot in the veins, but also in many cases to thickening in the walls and inflammatory thickening in the connective tissue immediately surrounding the veins. Periphlebitis is especially marked if the thrombosis is septic or purulent.

In cases of arterial occlusion the corresponding veins and venules are distended with black, fluid blood.

The consistence of the clot depends upon a number of factors, viz. the age (old or recent), and the aetiology of the process—

whether (a) simple aseptic propagation from a primary clot in some other part of the portal tract, or (b) secondary to phlebitis in the thrombosed vein itself, or (c) purulent.

A simple clot recently formed in a vein is nearly black in colour, elastic, non-adherent to the intima, easily withdrawn, and uniform in consistence. As the clot gets older, it loses its dark colour, till it becomes pale grey, firm and brittle, adherent to the vein-wall, and finally it may become organised and even canalised with fresh blood-channels¹.

In some cases where the thrombosis is due to local phlebitis the clot may be stratified², the outer layers being paler than the central core; a thrombus of this description is usually adherent to the intima from the first, and may retain a central channel unobstructed. The changes that take place with increasing age are similar to those in a simple clot.

A purulent thrombus may vary in its constitution; it may be composed almost entirely of pus transforming the lumen of the vein into an abscess-cavity, or of blackish soft gelatinous material containing pus-cells, or of semi-fluid blood mixed with pus³. For this reason it is impossible to draw a hard and fast line between purulent phlebitis and venous thrombosis.

DISTRIBUTION OF THE THROMBUS.

Venous thrombosis in the mesentery and intestine may remain strictly localised, or it may spread from one part into practically the whole of the portal tributaries⁴. Again it may take origin from the portal vein, and involve secondarily tributaries in different parts of the mesentery and intestine, producing several infarcts⁵. Finally the process may start in one tributary and involve tributaries at a distance by retrograde venous embolism through the trunk and intervening veins: this is especially the

¹ De Jong, No. 8.

² Schmorl (revd. by Amos), Frerichs (revd. by Borrmann), Letulle and Maygrier, *London Hosp. P. M.'s* 1908, No. 1144.

³ All these degrees in purulent thrombosis are well illustrated by Polya's four cases.

⁴ De Jong, No. 8, *London Hosp. P. M.'s* 1908, No. 1144, Mouchet (revd. by Zesas), Fedorowitsch, Kanis.

⁵ Welch and Flexner (revd. by Jackson, Porter and Quinby), Seitz (revd. by Borrmann).

case where thrombosis arises in the appendicular region and spreads into the veins of the jejunum¹.

As a general rule the venous radicles draining the intestine and the anastomosing arcades are thrombosed at a later date than the radiating larger tributaries of the superior mesenteric.

If we can depend upon the incidence of infarction of the intestine it appears that the jejunal and ileal veins are thrombosed with almost equal frequency. As we have seen above however (*vide* 'Mesenteric venous occlusion'), the venous thrombosis is generally far more extensive in any given case than can be gathered from the size of the infarcted area of intestine.

The following table gives an approximate idea of the distribution of mesenteric venous thrombosis:—

Jejunal veins	20 cases
Ileal veins	19 „
Both jejunal and ileal veins	23 „
?Jejunal, ?ileal veins	25 „
Right colic vein	7 „
Middle colic vein	4 „
Left colic vein	1 case
Veins of both small and large intestines	5 cases
Veins of ?small, ?large intestine	3 „

This table leaves out of account the involvement of the mesenteric trunks and portal vein (referred to above under 'Mesenteric venous occlusion').

(B) ARTERIES.

The alteration in the appearance of the mesenteric arteries by thrombosis is not so gross as in the case of the veins, though the arteries become firmer to the touch.

The obstruction may consist of an embolus blocking the mouth of the trunk or one of the main branches, or of a short thrombus², or of a thrombus completely occluding a considerable length of the trunk and branches³.

¹ This point is emphasised by Polya (*loc. cit.* S. 300).

² Jackson, Porter and Quinby, No. 18 (thrombus 2 cms. long), Trübel (revd. by Neutra).

³ Jackson, Porter and Quinby, No. 20, Watson (revd. by Neutra), Leclerc and Beutter: in the last case the trunk and origin of the large branches were thrombosed.

In most cases the artery and branches peripheral to the clot, both in cases of embolism and thrombosis, are quite empty of blood.

In cases of venous thrombosis the arteries are full of blood, and the capillaries distended.

Distribution of lesions resulting from mesenteric arterial occlusions :—

Infarction of jejunum only	6 cases
„ „ ileum only	24 „
„ „ jejunum and ileum	29 „
„ „ ?jejunum, ?ileum	16 „
„ „ ileum and colon	7 „
„ „ colon only	16 „
„ „ part of small and large intestines	22 „

The relative frequency of infarction in different parts of the colon is seen in the next table:—

Infarction of the ascending colon	43 cases
„ „ transverse colon	19 „
„ „ descending colon and sigmoid	7 „

Character of the clot. Recently formed thrombus is of a dull red-brown colour, elastic, non-adherent to the intima, and easily withdrawn. It is uniform in consistence, and not stratified. As it gets older it gradually loses its colour, till it assumes a pale reddish grey appearance, becomes adherent to the intima, dry, granular and friable.

Apart from the history of a case it is often impossible to say whether a clot is a thrombus or an embolus; not uncommonly it is both, the former being grafted on to the latter. An embolus generally lodges at a bifurcation of the artery¹, or opposite the offset of a branch partially or completely occluding one or both branches. The injury to the intima produced by the impact may account to some extent for the supervention of thrombosis.

¹ London Hosp. P. M.'s 1907, No. 1120, Moyes (revd. by Neutra).

(C) EFFECTS OF BLOOD-STASIS UPON THE CAPILLARIES AND VENULES.

Occlusion either of an artery or a vein in the mesentery leads to grave results in the capillaries and venules of the area affected if an adequate collateral circulation is not at once established.

With regard to venous obstruction, Lapointe (quoted by Mauclore and Jacoulet) says 'When insufficient collateral venous circulation is present the stagnant blood produces an engorgement with progressive tension which soon forms an insuperable barrier to the blood in the capillaries. The corresponding arteries continue to bring blood: it distends the capillaries, stagnates in turn, alters the vascular walls, and an infarct is produced.' The effect of an obliteration in an artery is the same, though brought about in a different way. The sudden removal of the *vis-a-tergo* leads to a retrograde venous engorgement, affecting the vessels as far back as the point of obstruction.

The immediate consequence of the raised blood-pressure combined with stasis and therefore carbonic acid poisoning, is irreparable damage to the delicate walls of the capillaries and venules which consist merely of a single layer of endothelial cells. Extravasation of serum and blood follows, leading, according to the severity of the lesion, to oedema, ecchymoses, haematoma, or haemorrhagic infarct and gangrene.

In a few cases¹ the process has been severe but localised, and has led to perforation from complete necrosis of the whole thickness of the wall of the intestine.

(2) MESENTERY AND GLANDS.

In the vast majority of cases gross vascular occlusions in the mesentery give rise to oedema in the affected area. This is one of the first changes and one which may advance to an extreme degree producing enormous thickening. The mesentery may measure anything from $\frac{1}{2}$ to $1\frac{1}{2}$ inches in cross section, and in one instance it measured 2 to 3 inches².

If the oedema is localised it may simulate a glandular

¹ Adénot (2 cases revd. by Neutra), Elliot.

² Jackson, Porter and Quinby, No. 27.

enlargement with periadenitis¹, the fat and connective tissue being firm, hard and inelastic.

With the increase in thickness there is a corresponding diminution in mobility of the mesentery, and this in itself may be a factor in the production of intestinal obstruction which so commonly follows in these cases.

Extravasations of blood are often found in the mesentery in the region of occluded vessels. They vary in size from small petechiae and ecchymoses to large collections of blood² capable of being felt through the abdominal wall³. In cases of arterial occlusion the haemorrhages are probably due entirely to venous back-flow and not, as it has been supposed by some authors⁴, to a collateral arterial supply.

The collateral arterial supply in the mesentery is generally very meagre, and the point has been emphasised by Taravellier⁵ that were the anastomosis responsible for the haemorrhages one would expect them to be most marked at the boundaries of the affected area. In actual fact the haemorrhages are most intense about the obstructed vessels where compensation is least.

As the process of infarction advances, the mesentery becomes darker in colour till it may be almost black, the serous coat loses its gloss and tears easily, and the fat, connective tissue and blood-vessels become necrotic.

The demarcation of the infarct from the healthy parts on either side may be sharply defined or gradual, whether the causal lesion is arterial or venous, or both arterial and venous together.

	Sharply defined	Ill defined	Sharply defined at one end and ill defined at the other
Arterial occlusion	30 cases	29 cases	2 cases
Venous occlusion	28 „	23 „	2 „
Arterial and venous occlusions together	4 „	5 „	0 case

¹ Original case No. 5.

² Cohn, Jastrowitz, Drozda (all revd. by Neutra), Riis (revd. by Jackson, Porter and Quinby), Thévenot and Rey.

³ The palpability through the abdominal wall of a haematoma in the mesentery is mentioned on the authority of Gerhard (quoted by Zesas), though it has not been possible to find any case in which this sign was elicited for certain. The cases referred to in a later section where a tumour was felt probably owed this sign to the infarcted coil of intestine.

⁴ Virchow, Cohnheim (quoted by Taravellier).

⁵ Taravellier, *loc. cit.* p. 18.

It might be expected that in cases of arterial occlusion the demarcation would be sharper than in those following venous obliteration, owing to the different capacities for compensation in the two systems, but in view of the uncertainty and scantiness of the data at our disposal no definite rule can yet be made. In all the certain cases of anaemic infarct the anaemic areas have been sharply defined.

In a few cases of septic venous thrombosis abscesses¹ have been found in the mesentery. In one case² of arterial thrombosis the mesentery contained gas vesicles along the line of the intestinal attachment, while in another³ they were present in this position and also beneath the intestinal mucosa.

The mesenteric lymphatic glands are frequently described as being much enlarged, and they often contain extravasations of blood. At later stages of the disease they undergo the same necrotic changes as those described above.

(3) INTESTINES AND THEIR CONTENTS.

There are two main types of lesion that are conditioned in the intestine by closure of its vessels,—(A) haemorrhagic infarction, and haemorrhagic processes of minor severity, and (B) anaemic gangrene or ischaemia of the intestine.

(A) HAEMORRHAGIC INFARCTION.

Haemorrhagic infarction and its allied conditions will be considered first as they constitute the vast proportion of the cases. As we have seen above, this class of lesion may be brought about by:—

(a) closure of the artery of supply by embolism, thrombosis, extreme endarteritis, atheroma, or extreme arterio-sclerosis.

(b) closure of the veins draining the part by thrombosis.

(c) simultaneous closure of both artery and vein by any of these means.

¹ London Hosp. P. M.'s 1910, No. 458, Polya, No. 1.

² Welch and Flexner (revd. by Jackson, Porter and Quinby).

³ Faber.

The extent of the infarct varies considerably in different cases. On the one hand there may be merely a few necrotic patches¹, or only a few centimetres² may be involved, while on the other the whole of the small intestine from the duodeno-jejunal flexure to the ileo-caecal valve³, or the whole of the small and large intestines⁴, is affected. The above extremes however are exceptional, the infarct generally extending for about 30 or 40 cms. Though infarction is confined as a rule to a single, continuous segment of intestine and mesentery, yet there are several cases⁵ on record where there have been two or more infarcts of a size varying from mere spots to coils of 15 or 30 cms. separated by engorged or fairly normal intestine. In one case⁶ the ileum, with the exception of the last 2 feet, was infarcted together with the ascending and transverse colon. The infarcts are not necessarily situated close together; in one case⁷ there were two in the mesentery while a third was found in the meso-sigmoid. As in the case of single infarcts the multiple ones may be sharply defined⁸ or their boundaries may be indistinct⁹.

Irregularity in the degree of infarction is attributed by Neutra to the irregularity in the collateral supply and to variability in the degree of thrombosis in terminal vessels. It is easy to see that if part of the blood can be drained by collaterals from a thrombosed venous area the engorgement will be less intense here than in those parts where the veins are completely thrombosed. In the same way the upper end of the jejunum and the middle of the transverse colon, which receive part of their blood-supply from the inferior pancreatico-duodenal and the inferior mesenteric respectively, are less likely than the intervening intestine to

¹ Prütz (revd. by Zesas), Osswald.

² Brunner, Burgess.

³ Ginsburg, Lilienthal (both revd. by Jackson, Porter and Quinby), Lobert, Lothrop (revd. by Neutra), Köster, Sachs (revd. by Kölbing), Johst (revd. by Maclaure and Jacoulet), de Jong, No. 8.

⁴ Munro, Westhoff.

⁵ Borrmann, No. 1 in Jackson, Porter and Quinby's series, Munro, Osswald, Seitz (revd. by Borrmann), Parensky (revd. by Neutra), Lagane, No. 5.

⁶ No. 1 in Jackson, Porter and Quinby's series.

⁷ Munro.

⁸ Parensky.

⁹ Lagane, No. 5, Osswald.

undergo infarction if the superior mesenteric arterial trunk is closed¹.

The condition of an infarcted coil resembles very closely that in a strangulated hernia, as would be expected from the similarity in the state of the vessels in the two cases. Further, a coil in which the circulation has been impaired by embolism or thrombosis of its vessels displays all the variability of a strangulated coil according to the degree of the vascular obstruction: thus there may be merely oedema and engorgement of the mesentery and intestine, or infarction, or finally gangrene.

An infarcted coil is dark red, purple or blue-black, and is occasionally mottled with green patches; it is usually much thickened² from infiltration of its walls with blood and serum, and distended from accumulation of gas in the interior. The serous coat often retains its gloss till a late stage; it may be covered with shreds of fibrin, binding the affected segment to the abdominal wall or to adjacent coils of intestine. The mobility of the affected coil is either seriously impaired, or totally absent, especially if peritonitis is present³.

When the wall of the infarcted coil is cut through it is seen to be much thickened, black, oedematous, and engorged with blood in all its coats. Either the whole wall or only the mucosa may be necrotic; the latter is often separated from the rest of the wall, and is sometimes entirely absent in patches. The mucosa may be covered by a false or 'diphtheritic' membrane⁴.

Perforation from necrosis of the whole thickness of the wall has been reported in several cases. The affected coil after perforation is of course no longer distended, but flaccid and inert.

Out of eight cases in which the superior mesenteric vessels were occluded the perforation took place in the ileum (generally near

¹ This rule does not apply to closure of branches of the superior mesenteric artery: the anastomoses between individual branches in the middle of the area of supply are richer than those between outlying branches and the inferior pancreatico-duodenal above, and the inferior mesenteric below.

² Exceptional cases were reported by Munro, and Jackson, Porter and Quinby (No. 3).

³ The factors determining immobility in an infarcted coil will be considered under 'Symptomatology.'

⁴ Lagane, No. 5, Talke, Monro and Workman, Lobert (revd. by Neutra).

the lower end) in seven¹, and in the transverse colon in one². The vascular obstruction was situated in the artery in six cases, and possibly also in the remaining two³. We have already seen that perforation of the descending colon was found in three cases of occlusion of the inferior mesenteric artery.

Contents of the Infarcted Coil.

The infarcted intestine generally contains dark, offensive, semi-fluid material which is either streaked with blood, or consists of almost pure blood. The latter may be partly clotted or of tarry consistence, or it may be dark red and liquid. Free blood may be present in the lumen of the intestine in cases where none has been passed per rectum or been seen in the vomited matter; this is probably due to local paralysis of the intestine at the site of the infarct. In the infarcted coil there is generally an accumulation of gas, which in the normal state would have been absorbed by the veins. Kader has shewn that the gas accumulation is directly dependent upon circulatory disturbances in the intestine. His classical series of experiments will be referred to later.

Condition of the Coils above and below the Infarct.

Apart from experiments in which careful observations have been taken it is impossible to collect sufficiently accurate data to formulate any definite rule regarding the condition of the coils immediately above and below the infarct; but so far as we know the afferent coils are generally rather more distended than the normal, though not so distended as the infarcted coil, while the efferent coils tend to be somewhat contracted.

In those cases where there was no clear line of demarcation, the afferent and efferent coils shewed signs that are associated with the earlier stages of the disease, viz. oedema, engorgement and haemorrhages in varying degrees.

¹ Blake, Fischer (revd. by Mauclaire and Jacoulet), Jackson, Porter and Quinby, No. 18, Kaufmann, Kiliani, M'Carthy, London Hosp. P. M.'s 1908, No. 1230.

² Conti (revd. by Neutra).

³ London Hosp. case referred to above (1), and Blake.

Morbid Histology.

The first and mildest change that takes place in the intestine after closure of its artery or vein consists of oedema and leucocytic infiltration. Lagane says that the latter consists mainly of mononuclear cells, but that there are a certain number of polynuclear eosinophiles. Minute capillary haemorrhages that sometimes occur at this stage are always discrete. The tissues at this stage are very much thickened on account of the infiltration with serum and leucocytes. It has been shewn experimentally¹ that if recovery is allowed to take place at this stage, stenosis of the intestine may follow.

The second stage of the disease is that of engorgement. The capillaries and venules become enormously distended with blood, which however is as yet extravasated to only a trifling extent. The intestine is still viable (an important clinical point).

Haemorrhagic infarction constitutes the next stage. The intestine shews enormous engorgement and extravasation of blood into the coats, especially marked in the sub-mucous layer, and to a less extent beneath the peritoneum. In the sub-mucosa the blood forms 'vast lakes' which may 'completely separate the mucous and muscularis coats' (Lagane). Necrotic changes are most marked in the mucosa: here the villi are either completely degenerated and full of blood, or are absent entirely, and there is extreme leucocytic infiltration of the underlying portion of the sub-mucosa. Lieberkühn's glands are distended and partly necrotic, but according to Frattin (quoted by Mauclaire and Jacoulet) it is from these that the epithelium may be regenerated. The muscularis mucosae is engorged with oedema and extravasated blood. The muscular coat shews less changes than any, though the fibres are dissociated by haemorrhages (Lagane) and shew degenerative changes: the nuclei fail to stain, or are fragmented. Haemorrhages are present beneath the serous coat, which itself shews leucocytic infiltration.

It may be noted here that Lagane draws a distinction between haemorrhagic infarction, and what he calls 'apoplexy' of the

¹ Bolognesi.

intestine, the latter being a more advanced condition than the former. To quote his own words¹:—

‘ Dans l’infarctus, il y a envahissement, surréplétion des mailles des tissus normaux par les éléments du sang. Les tissus sont surdistendus par le sang, atrophiés par lui, mais le contiennent à la façon d’une éponge retenant un liquide. Dans l’apoplexie, au contraire, il y a remplacement de tous les éléments d’un tissu par le sang coagulé. L’épanchement hémorragique a tout repoussé, ou tout détruit, et ce n’est qu’un amas de sang que l’on examine.’

When the intestine has been infarcted from arterial obliteration, the veins in its walls are distended with fluid blood and leucocytes, the latter occupying the marginal part of the vessels (Lagane). There may be secondary thickening of the endo-vein. Around the veins there is often a zone of leucocytic infiltration. The venules and capillaries are enormously distended with blood which finally bursts through into the tissue-spaces; but the arterioles commonly shew no change of structure, though sometimes filled with fibrinous clot.

In infarction due to venous thrombosis the venules are filled with blood-clot, and their walls often shew definite inflammatory thickening. The tissue changes are practically the same as in cases of arterial obliteration, viz. loss of epithelium, oedema of the sub-mucous connective tissue, and atrophy of the gland cells: all are associated with enormous haemorrhagic effusion.

If the process of infarction advances further before the death of the patient, ‘apoplexy’ of the affected coil (Lagane) supervenes, and finally gangrene. The last is characterised by the invasion of the intestinal wall by septic or putrefactive organisms from the lumen of the bowel, and the walls already replaced to a large extent by blood-clot cease to cohere, and are easily perforated.

(B) ANAEMIC GANGRENE.

This condition was first obtained experimentally by Litten in 1875, but it was not until 1902 that the first case was described by Sprengel. Since then cases of a similar nature have been reported by Niederstein, Grawitz, Neumann, Zimmermann, Roper,

¹ Lagane, *loc. cit.* (thèse de Paris), p. 66.

Leclerc and Cotte, and Meyer; one original case is reported in this series. Within recent years anaemic gangrene of the intestine has been produced in animals by Kader, Niederstein, and Bolognesi.

No satisfactory explanation has yet been given for the occurrence of anaemic gangrene. The original hypothesis put forward by Sprengel that it was due to simultaneous obliteration of both artery and vein is inadequate, for the reason that out of 22 cases of double vascular lesions only three shewed anaemic infarcts, while out of the nine cases of anaemic infarct on record six would remain unexplained, shewing as they did a single vascular lesion instead of a double one.

Leclerc and Cotte in their work¹ upon anaemic gangrene of the intestine came to the following conclusion:—‘On pourrait dire que dans tous les cas où l’oblitération vasculaire mésentérique siège au-dessous des arcades anastomotiques situées dans la mésentère, elle donne lieu à un infarctus blanc anémique de l’intestin; lorsqu’elle intéresse seulement les grosses branches de la mésentérique elle déterminerait, au contraire, un infarctus hémorragique.’ This view is probably as accurate an explanation as can be arrived at up to the present, and it is one that is supported by the results of Niederstein’s experiments. On the other hand it does not cover Neumann’s case, that of Grawitz, nor the rather atypical one reported by Roper. Complete obliteration of the terminal arterioles does not appear to be necessary, so long as the blood-supply is cut down to a minimum. Chronic sclerosing processes in the arteries or arterioles were described in four of the cases². Now, as a result of chronic endarteritis or arterio-sclerosis the blood-supply to the affected intestine must have been steadily reduced; further, as a result of this it seems reasonable to suppose that the veins draining the part became reduced in size, and possibly in number, so that the venous regurgitation resulting from the removal of the *vis-a-tergo* was almost negligible. This view is offered as a supplementary explanation to that given by Leclerc and Cotte.

¹ Leclerc and Cotte, *loc. cit.* p. 496.

² Leclerc and Cotte, Zimmermann (revd. by Talke), Meyer, original case No. 1.

The nine cases of anaemic infarction may be briefly tabulated :—

Sprengel.	Anaemic gangrene of 10 cms. lower ileum.	No bleeding during resection: probable occlusion of small arts. and veins.
Niederstein.	Anaemic gangrene of caecum: engorgement of lower ileum.	Thrombosis of large part of sup. mes. art.: thrombosis of small veins draining caecum.
Neumann (revd. by Sauv�).	Anaemic gangrene of 25 cms. upper jejunum. Mucosa thickened.	Thrombosis of small branches of sup. mes. art.
Leclerc and Cotte.	Anaemic gangrene of 1 metre lower ileum.	No bleeding: marked endarteritis: veins thrombosed.
Zimmermann (revd. by Talke).	150 anaemic gangrenous ulcers in ileum.	Great narrowing of small arts.: outer coat much thickened.
Roper.	Small intestine distended and anaemic.	Embolism of sup. mes. art. below origin of middle colic.
Meyer.	Anaemic infarction of 4 or 5 cms. ileum. Mucosa thickened.	Endarteritis and thrombosis of small arts. and venules.
Grawitz.	Anaemic infarction of 25 cms. lower jejunum: afferent and efferent coils dark red.	Numerous emboli in larger branches of artery supplying infarct.
Original, No. 1.	Anaemic infarcts in 9 ins. lower ileum.	Thrombosis of corresponding small arts. Thickening of the walls of the anastomosing arcades of the affected area.

The wall of the intestine at the site of an anaemic infarct is white or yellowish grey in colour, and is entirely devoid of blood. At one or both ends of the infarcted segment there may be a demarcation zone of hyperaemia separating it from healthy intestine¹, as in the case of anaemic infarcts in other parts of the body. The wall of the affected coil is generally very thin; in Niederstein's case it was the 'thickness of a piece of paper.' In two cases² however it was thicker than the normal. The differences in

¹ Meyer.

² Neumann (revd. by Sauv ), Meyer.

thickness are attributed by Leclerc and Cotte to the varying intensity of the lesion.

There is nothing characteristic about the contents of the affected segment except the absence of blood. Blood may be present if the lesion is associated with much engorgement of the afferent and efferent coils, as in the cases reported by Grawitz and Niederstein¹.

(4) PERITONEUM.

When the general peritoneal cavity is opened in a case of haemorrhagic infarction or engorgement of the intestine it is common to meet with a considerable quantity of free blood or blood-stained fluid that has been extravasated from the mesentery and intestine. The blood may be partly clotted, or in the presence of peritonitis it is sometimes mixed with pus.

Blood or blood-stained fluid in the peritoneal cavity was reported in 37 cases of occlusion of the mesenteric arteries, 42 of mesenteric venous occlusion, and 6 of coexistent arterial and venous occlusion. In four cases² of venous thrombosis it measured 1 litre, and in one³ 1½ litres.

Ascites has been described in a good many cases (arterial 12, venous 16, and both arterial and venous 2) but in some of these it is possible it was not caused by the infarction but by the associated cardiac or hepatic disease.

The frequency with which peritonitis is found after obstruction in the mesenteric circulation demonstrates very clearly that it is not necessarily dependent upon perforation of the intestine, for as we have seen already perforation is rare in cases of infarction.

The incidence of peritonitis is given in the following table:—

	'Fibrinous' Peritonitis	'Fibrino- purulent' Peritonitis	'Purulent' Peritonitis	'Peritonitis'
Arterial occlusion	15 cases	3	8	19; also 1 faecal and 1 local
Venous occlusion	14 cases	3	6; also 2 local	12; also 1 faecal
Arterial and venous occlusion together	2; also 1 local		1; also 1 local	

¹ Leclerc and Cotte's case shewed haemorrhagic infarction of 1 metre of intestine above the anaemic infarct.

² Barth (revd. by Jackson, Porter and Quinby), Mauclair and Jacoulet, Solieri (revd. by the last named authors), Sprengel.

³ Askanaazy.

There is often free gas in the peritoneal cavity, the contents of which are generally described as being extremely offensive or of foetid ('aashaft') odour.

(5) BACTERIOLOGY.

Very little is known about the bacteriology of mesenteric vascular disturbances. In certain cases of descending venous thrombosis the process was indirectly due to syphilis, but the specific organism probably had nothing to do with the actual thrombosis. The same statement may be made with regard to the organism of acute rheumatism, which so commonly gives rise to endocarditis. Embolism following endocarditis is very common, but no cases of mesenteric embolism have been reported in which the organism has been isolated.

One case of venous thrombosis and two of arterial embolism were associated with streptococcal septicaemia. In another case, Gram-negative diplo-cocci supposed to be a variety of *Micrococcus catarrhalis* were grown from the blood of a patient who developed mesenteric embolism while in the London Hospital.

Finally, in Welch and Flexner's case the *B. aerogenes capsulatus* was responsible for the development of gas vesicles in the mesentery.

It is of interest that cultures taken from the veins after experimental ligature of vessels were found by Bolognesi to be negative. Identical results were obtained in cultures taken from the walls of the strangulated coils.

SECTION IV

EXPERIMENTS ON ANIMALS

(1) EXPERIMENTAL CLOSURE OF ARTERIES

NUMEROUS series of experiments have been carried out by different authorities for the purpose of reconciling the clinical results of occlusion of the superior mesenteric artery with the existence of an anatomically free system of compensatory anastomosing channels. Clinically, obstruction in the superior mesenteric artery is, with few exceptions, followed by infarction of the intestine. Anatomically the superior mesenteric anastomoses with the inferior pancreaticoduodenal, the last dorsal on the right side, the retroperitoneal plexus of Turner, and the inferior mesenteric. The last anastomoses with the middle and inferior haemorrhoidals.

The first experimental work was done by Litten in 1875. He chose dogs, rabbits and guinea-pigs for his experiments, and observed that ligation of the superior mesenteric trunk was always fatal in from 12 to 48 hours. The symptoms were:—great lassitude, extreme depression of the nervous system, anorexia, occasional vomiting; melaena with stools of foetid odour; then abdominal distension accompanied by subnormal temperature, and death. After death decomposition supervened rapidly.

Post mortem examination. In some animals purulent and haemorrhagic peritonitis occurred; in all, the mesenteric glands were enlarged, and in some, Peyer's patches also. From the duodeno-jejunal flexure to the upper part of the large intestine the gut was distended, dark black-red, flabby, lustreless, and engorged with sanious oedema: the mucosa was the same colour, was necrotic and shed a haemorrhagic exudate: the serosa was raised in vesicles ('blasig') in places. Large haemorrhages were present between the intestinal coats: the mesentery of the affected part was lustreless, contained innumerable ecchymoses, some of

which followed the course of vessels: some glands were infiltrated with blood. The veins were much distended with fluid blood down to the smallest tributaries, and there was no trace of clotting: coils of vessels in the villi were distended as though injected: some villi were swollen and some necrotic. Haemorrhages into Peyer's patches were less intense than into the glands. In short, there was necrosis with oedema and haemorrhages: 'disturbances of nutrition had led to haemorrhagic infarction.'

Ligature of a branch of the superior mesenteric led to the same changes in the corresponding segment of intestine and mesentery.

No movement was observed in the blood in branches below the ligature, either directly after, or after an interval of 12 to 14 hours. The process of infarction was observed microscopically in a few segments, and was attributed to venous back-flow.

Litten's control experiments. (1) After ligature of the superior mesenteric artery the latter was divided peripherally, but no blood escaped. (2) After ligature of the artery the intestine was not coloured by injection of sodium sulphindigotate (50 to 200 c.c.) into the jugular vein or heart in live dogs. The rest of the body was stained a rich blue colour.

Failure of the anastomoses was not due to a fall in blood-pressure: the latter was higher by 20 to 25 mm. Hg. in the carotid after ligature than before.

Forced injections.

The superior mesenteric was tied in a medium sized dog, and a cannula connected with a manometer tied into the carotid. Indigo solution was introduced under pressure through the cannula: at 120 to 130 mm. Hg. pressure the solution did not penetrate the 'infarct' area, and only at 300 mm. Hg. did it begin to reach the larger arteries of the gut without filling the smaller arteries or capillaries: the same happened at 350 mm. Hg. pressure. Thus the resistance of the area supplied by the superior mesenteric was far too great to be filled through any collaterals under normal physiological pressure.

Litten's conclusion therefore was that the superior mesenteric was an 'end-artery' physiologically, though anatomically 'anastomosing.'

Faber in the same year gave the following explanation for the failure in the collateral supply through the inferior pancreaticoduodenal and inferior mesenteric arteries after closure of the superior mesenteric. To begin with, the inferior vessel is much smaller than the superior, and therefore cannot assume the functions of the latter suddenly. Again, the length of the anastomosing arcades to be traversed by blood from the inferior mesenteric is extremely long (from the duodeno-jejunal flexure to the middle of the transverse colon) and the natural channel through the inferior mesenteric and gastro-duodenal is much easier and shorter. Finally, the blood-pressure in the portal vein is lowered as a result of the removal of the normal *vis-a-tergo* supplied by the occluded superior mesenteric artery, and the return-flow of blood through the inferior mesenteric vein is therefore rendered easier than before. The normal course for the blood in the inferior mesenteric artery consequently is made easier than through the anastomoses into the superior mesenteric system.

Bier, in 1897, endeavoured to shew by a series of experiments that the absence of a collateral circulation was due to a deficiency in the 'sensation of blood' (*Blutgefühl*) in the intestine. As a result of this deficiency there are, according to his view, no afferent impulses when the blood-supply is cut off or lowered, and therefore no reflex vaso-dilatation. In his opinion the circulation in the intestine is regulated by the stimulus afforded by the intestinal contents.

In reference to this theory Neutra remarks that an absence of '*Blutgefühl*' in the small intestine is not remarkable when it is remembered that the latter is also anaesthetic to pain and heat. Orthner is of the opinion that Bier failed to supply evidence that the part of the large intestine supplied by the inferior mesenteric had any better '*Blutgefühl*' than the rest of the intestine, large or small. Orthner favours the simpler explanation of Litten and Faber.

Bier's hypothesis is difficult to reconcile with those exceptional cases in which a collateral circulation was established in the superior mesenteric area, for according to it there should be no stimulus for the production of any compensatory supply, and no

reflex vaso-dilatation. Such cases are explained by Neutra upon the assumption of a congenitally rich system of anastomosing channels, but even upon this hypothesis it is difficult to understand how a collateral circulation could have been established unless the closure had been a very gradual one, as in Virchow's case and case No. 6 (original) of this series. In this connection it is of interest that Longhope and McClintock¹ succeeded in producing a complete collateral circulation in the mesentery of dogs by narrowing the lumen of the superior mesenteric artery by means of aluminium bands or Halstead's constrictors. In one of the animals the superior mesenteric was found after 5 months to have been converted into a fibrous cord. In some of the dogs in which thrombosis took place soon after the operation, hæmorrhagic infarction followed.

Other observers who have repeated Litten's experiments have obtained fairly constant results after ligaturing the trunk of the superior mesenteric artery. The effects of ligaturing branches have on the other hand led to wide difference of opinion as to whether the superior mesenteric is an 'end-artery' or not. Lagane is of the opinion that a single artery and a single vein are capable of providing for the circulation of a segment of intestine measuring 50 cms., on condition that the rest of the vessels corresponding with this segment are ligatured at sufficiently long intervals of time to permit of adaptation in the collateral channels.

He therefore considers the superior mesenteric artery a functionally anastomosing vessel by virtue of its arcades, and in this view he is in agreement with Frattin². Lagane found that isolated ligature of arteries or veins corresponding with 20 or 30 cms. of intestine at a distance of 2 cms. from its mesenteric attachment was followed by no infarction or necrosis; but that ligature of the arteries of a segment of 30 cms. of small intestine 6 cms. from its attachment to the mesentery, after injection of *B. typhosus* into the affected segment, led to necrosis.

Bolognesi summarises the views held by different observers who ligatured branches of the artery. Thus, Cohnheim, and Niederstein obtained no changes, Tangl and Harley, and Kader found infarction; Maas, necrosis of the gut-wall followed either by

¹ Quoted by Collins.

² Quoted by Lagane.

early death or by stenosis of the intestine; Clarrin and Monier, Vinard, degenerative changes in the corresponding segment; and Ravenna, no change or else haemorrhagic infarction.

Bolognesi attributes the variability in the results from ligaturing vessels to the difference in arrangement of experiments, the kind of animals used, the position of the obstruction, and the physiological condition of the gut at the time of operation. He concludes by saying that between complete collateral compensation and its absence with necrosis of the gut, there is a third possible result, viz. the production of oedema limited to the sub-serosa and sub-mucosa, thickening of tissue and atrophy of the epithelium: and that these changes may be followed by contraction of the gut and stenosis.

Far greater unanimity exists upon the effects of experimental embolism of twigs of the superior mesenteric artery. Lagane remarks that experimental embolism is equivalent to the placing of an infinite number of ligatures upon arteries and veins: in the veins the emboli prevent removal of blood through collaterals, thus increasing the (capillary) tension; while in the arteries they prevent blood from reaching the part by direct or by collateral channels. The results are diapedesis from stasis, rupture of the capillaries, and retrograde flow of blood, all of which lead to infarction.

Once more, to quote Bolognesi's summary of the results obtained by different authors, Panum, Lefeuvre, and Felz obtained haemorrhages and ulcerations from experimental embolism; Marek, Niederstein, and Poddigha, necrosis and gangrene; Ravenna, haemorrhagic gangrene; and Cohnheim alone, no positive results.

Experimental injections of wax and other emulsions are of interest from the evidence they afford of the relationship between mesenteric embolism and purpura. Talke in his endeavour to prove this relationship quotes Panum's work, a brief summary of which may be given.

Panum injected a fine emulsion of black wax or air into the femoral artery of dogs. A few hours after the operation he observed the development of paralysis, respiratory changes, and gastro-intestinal disturbances such as nausea, vomiting of bilious or sanious material, and evacuations of blood from the bowel.

After injection of air, these phenomena were somewhat less marked than after wax injection, but both series produced cutaneous petechiae and ecchymoses, intensely injurious effects upon the walls of the gastro-intestinal tract, and embolism of the larger and smaller arterial branches in the intestinal walls. The mucosa of the stomach and intestine shewed numerous ecchymoses from the size of a grain of sand to that of a hempseed, irregular in outline and dark red in colour: the arterioles supplying these spots were blocked by small particles of wax. Similar spots were also found in the serosa of the intestine, and had been produced in the same way. There were small circumscribed haemorrhagic infarcts involving the whole thickness of the intestinal wall. A large amount of blood-stained mucus was present in the stomach and intestine. In some places ulcerations were found extending down to the muscular coat: these were produced in from 9 to 22 hours, the arterioles of supply being found plugged with the wax injected. No wax was found in vessels supplying healthy mucous membrane. In the stomach, large, black, yellowish grey or brownish ulcers were present, similar to those in the small intestine: the muscular coat beneath them was infiltrated with wax, and the arterioles blocked with wax emboli.

Talke, commenting upon Panum's experiments, says that they are of significance in that they prove the concurrent production of cutaneous and intestinal lesions by embolism.

(2) EXPERIMENTAL CLOSURE OF VEINS.

Infarction or other serious damage to the intestine is not nearly so likely to occur after closure (experimental or otherwise) of the superior mesenteric vein as after closure of the corresponding artery. Cases have been mentioned above¹ in which the trunk of the vein had been tied near its termination without producing adverse clinical results.

Solowieff in 1875 tied the superior mesenteric vein immediately below its junction with the splenic in dogs, and the animals survived for weeks or even months. The same results followed ligation of the splenic.

¹ Mayo Robson, Wilms.

Kader, in 1892, in the course of investigations upon gas accumulation in the intestine, applied moderately firm ligatures to the root of the mesentery, thereby compressing the veins without the arteries. This procedure was followed by the following changes:—Profound changes in the walls of the intestine, oedema, and infiltration with blood: great thickening of the walls, all the greater if the hyperaemia was progressive: after a few hours, complete paralysis of the muscular coat. In the intestine, besides the original contents there were extravasations of blood, and much gas: the longer the duration of the hyperaemia the greater was the accumulation of gas: the segment became tightly stretched, and its circumference doubled or trebled. Haemorrhagic gangrene of the affected coil supervened, and perforations (minute and numerous) sometimes occurred at the mesenteric attachment of the intestine, leading to the escape of gas. The boundaries of the infarcted coil were sharply defined. In the peritoneal cavity there was a sanious exudate.

The above results occurred only if the collateral circulation did not at once suffice to adjust the disturbances in the strangulated coil.

Bier, who used dogs, pigs and guinea-pigs, never saw a trace of hyperaemia after even firm strangulation in from 2 to 20 minutes if the coils affected were empty; if, however, the coils were full, ligature of the mesentery produced marked hyperaemia in 10 minutes.

Kader, by further experiments, demonstrated that distension of the intestine with gas was directly dependent upon failure of absorption and removal of the gas through the veins, and not, except to a very trifling degree, to the presence of much or little material in the intestine.

Wilms (quoted by Zesas) invariably obtained haemorrhagic infarction by ligaturing the trunk of the superior mesenteric vein in rabbits.

Ravenna (quoted by Bolognesi) and Niederstein obtained haemorrhagic infarction in the same way, but the latter found no marked changes after ligaturing tributaries, unless he tied the collaterals as well; in this case haemorrhagic infarction followed. Similar results were reported by Frattin (quoted by Mauclair and Jacoulet).

Béguin (quoted by Mauclaire and Jacoulet), experimenting on dogs, shewed that an anastomosing venous arcade was only capable by itself of draining blood from a segment of less than 30 cms. of intestine, and that the intra-parietal anastomosing venules were able to drain less than 5 cms. if the anastomosing arcades were isolated from the intestine: if these limits were exceeded gangrene followed. These results have been supported, except as regards minor details, by a number of other observers (Ravenna, Bier, Madelung—quoted by Lagane).

Ravenna (quoted by Bolognesi) never observed infarction of the colon after ligature of the inferior mesenteric vein, unless tributaries of the superior mesenteric had been tied as well.

When the difference in the results obtained by different observers is considered, some allowance must be made, as in the case of the experimental work on the arteries, for difference of technique, the kinds of animals used, etc. As a general rule it appears that haemorrhagic infarction follows ligature of the trunk of the vein or of the artery, but that it is more prone to occur after the latter than the former operation: further, that ligature of medium sized tributaries of the vein, except in large numbers, commonly leads to no gross changes in the intestine; but that ligature of a considerable number of venous radicles close to the line of attachment of the intestine generally leads to infarction.

(3) EXPERIMENTAL CLOSURE OF ARTERIES AND VEINS TOGETHER.

Simultaneous obstruction in branches of the superior mesenteric artery and in tributaries of the corresponding vein was obtained by Kader by means of a firm ligature applied to the mesentery. The following changes were observed:—Pallor and at the same time cyanosis of the strangulated coil followed directly after: the colour changed later to dark ashy grey, or yellowish grey. The vessels first appeared as a blue-black network. No transudation occurred into the lumen of the intestine or into the peritoneal cavity. Sanious fluid in the peritoneal cavity appeared only if peritonitis was present. There was gas accumulation in the strangulated coil, and it was favoured by the presence of intestinal contents, though the latter were not essential. Distension of the

coil was not so great as after moderate strangulation (i.e. venous occlusion alone). The gut-walls became thin and flabby, and constant early perforation took place. 'Bead-like' (perlschnurartig) contractions in the affected coil were well marked at first, but soon ceased. The whole course was more acute than after moderate strangulation.

Lagane after tying both arteries and veins corresponding with a segment of 25 cms. of small intestine and at a distance of 2 or 3 cms. from its attachment failed to obtain any lesion: the animal (a sheep-dog) after the operation merely shewed abundant 'serous' diarrhoea. After the same procedure had been carried out in a segment of 35 cms. at a distance of 4 cms. from the intestinal attachment, infarction and gangrene followed in 24 hours.

The same author remarks that after ligature of tributaries of the vein, the addition of an arterial obstruction (whether by artificial emboli or ligatures), unless involving a very large area, has but little effect upon the character of the lesion: he found that local ischaemia was prevented in these cases by the collateral arterial supply.

Marek, in 1907, obtained anaemic infarction by closure of the artery and vein simultaneously. He injected the veins of the segment chosen with indigo-carmin solution, thereby preventing any venous back-flow; then he tied the artery of supply. Under these conditions anaemia and necrosis followed, whether all collaterals were cut off or not.

Niederstein agrees with Marek as to the necessity of simultaneous closure of arteries and veins in the production of anaemic infarction, but while admitting the possibility of a venous back-flow emphasises the importance of cutting off all collateral arterial channels. He says that if this is not done haemorrhagic infarction follows.

The objection to the view that the blood in haemorrhagic infarction comes from collateral arterial channels has already been discussed (*vide* 'Morbid Anatomy').

Cohnheim (quoted by Bolognesi) failed to obtain haemorrhagic infarction by ligaturing the artery alone, but observed it after simultaneous ligature of both artery and vein. Ravenna (quoted

by Bolognesi) obtained the same results as Cohnheim after tying both artery and vein together.

Separation of the mesentery from the intestine close to the line of attachment, a procedure that amounts to the same thing as cutting off the arteries and veins, leads in the great proportion of instances to gangrene of the segment of intestine thus shut off (Madelung, Rydygier, Tansini—quoted by Bolognesi—Bier, etc.). According to Zesas, however, a separation of the mesentery from the intestine proximally to the anastomosing arcades can be carried out for a considerable distance without producing any harm to the bowel.

*Experimental resections¹ of extensive segments
of Small Intestine.*

Flint carried out experiments upon 14 dogs to determine the effect of extensive resections of the small intestine upon nutrition. 'Of the 16 operations upon 14 dogs, 6 died from all causes, making a total mortality of 37 %.' He says that it is difficult to say the maximum amount that can be removed without danger to life, as so much depends upon the general condition and age of the animal at the time of operation. Young animals do not stand extensive resections so well as older ones.

To quote his own words:—'This much can be said: dogs from which about 80 % of the combined ileum and jejunum have been removed, or short circuited (Dogs Nos. 1, 9, 10 and 12) may live indefinitely after the operation.'

The first effects of the operation are profuse diarrhoea and loss of weight, from which the animal slowly recovers: but though the animal becomes apparently normal, it is very sensitive to dietetic disturbances, and any conditions of severity may cause death from marasmus or enteritis.

In three of the animals in which only 65 % of the total intestine had been removed a ravenous appetite followed in spite of an unlimited diet, and they never recovered their normal condition.

¹ Although these experiments were of a totally different nature from those described above, and were concerned entirely with the operative treatment of the disease, yet it will be more convenient to mention them here than in the section dealing with treatment.

If an easily absorbable diet was given to the animals after operation, there was no increase in the amount of fat or nitrogenous material in the faeces; but if the fat in the diet was increased, both fat and nitrogen in the faeces at once increased, reaching in some instances one fourth of that ingested.

The urine of animals with shortened intestines shewed an absolute increase of conjugated sulphates, and a relative increase of alkaline sulphates, indicating that there was an excess of intestinal putrefaction.

SECTION V

DESCRIPTIVE ANATOMY

It will not be unprofitable at this stage to consider briefly the anatomical distribution of the vessels of the mesentery and meso-colon, and the collateral channels whereby blood may reach and leave the intestine when the main vessels are obstructed.

(1) THE MESENTERIC ARTERIES.

(1) The superior mesenteric artery supplies the whole of the small intestine from the duodeno-jejunal flexure to the ileo-caecal valve, together with the caecum, the ascending and first half of the transverse colon. It assists the inferior mesenteric in the supply of the second or left half of the transverse colon.

The branches of the superior mesenteric are the *rami intestini tenuis*, the ileo-colic, the right colic and the middle colic: these form an anatomically rich system of anastomoses within the mesentery and meso-colon by dividing and joining again with each other into primary arcades. These in turn divide and anastomose into three or four tiers of arcades, and from the most distal of these small twigs radiate to enter the wall of the intestine, where they form further anastomoses with each other.

The above description holds good for the greater part of the superior mesenteric, but it has been shewn by Lardennois and Okinczyc (quoted by Sauv ) that at the two extremities of the system there are neither any anastomosing arcades nor any significant communications with the coeliac axis above nor the inferior mesenteric below. Thus obliteration of a large branch at either end of the area of supply is of vastly greater significance than that of one near the middle.

(2) The inferior mesenteric artery is a much smaller vessel than the superior. It supplies the second half of the transverse colon, together with the descending, iliac and pelvic colon, by dividing up into the following branches:—The left colic, two or more sigmoid branches, and the superior haemorrhoidal which is the terminal part of the trunk.

The number of anastomoses between the branches is much smaller, and the bowel is mainly dependent upon a marginal vessel derived from the ascending and descending branches of the left colic, and the sigmoids (Manasse). The marginal vessel sends off twigs close to the intestinal attachment to the meso-colon.

(3) *Anastomoses between the mesenteric and other arteries.* The communications between the mesenteric arteries and others mentioned below should not be reckoned as functionally capable by themselves of supplying enough blood for the vitality of the intestine, though they may become so under certain circumstances which are very rare.

(a) The inferior phrenic with the upper jejunal arteries through the hepatic, gastro-duodenal, superior pancreatico-duodenal and inferior pancreatico-duodenal.

(b) The lumbar arteries (4 pairs); small branches anastomose with branches from the hepatic and colic arteries to form the retroperitoneal plexus of Turner.

(c) The last dorsal and the ileo-colic.

(d) The internal iliac with the inferior mesenteric through the internal pudic, inferior haemorrhoidal, and middle haemorrhoidal, the latter itself arising from the anterior division of the internal iliac.

(2) THE MESENTERIC VEINS.

The distribution of the veins in the mesentery and meso-colon corresponds to a great extent with that of the arteries, and it will not be necessary to devote much space to a consideration of the veins themselves.

The portal system differs from the caval venous systems in that it is situated between two capillary systems—that of its drainage area, and that of the liver. Further, the veins constituting it are devoid of valves.

(1) The superior mesenteric drains the whole of the small intestine with the exception of the duodenum, the caecum, the ascending and first half or two thirds of the transverse colon. It joins the splenic to form the portal vein.

(2) The inferior mesenteric drains part of the transverse colon, the descending colon and sigmoid, and the upper part of the rectum. It enters the splenic close to the termination of the latter.

(3) The anastomoses between the tributaries of the mesenterics are much freer than those of the branches of the mesenteric arteries. From the point of view of morbid anatomy, as well as of clinical medicine, the anastomoses between the portal and caval systems are of great importance. De Jong has given a very detailed description of them, and the following summary has been made from his work.

The following 'accessory veins of Sappey' which normally drain blood into the liver form anastomoses with the systemic circulation :—

- (a) Tributaries from the gastro-hepatic omentum.
- (b) " " wall of the gall-bladder.
- (c) " " Glisson's capsule.
- (d) " " the suspensory ligament.
- (e) Para-umbilical veins : occasionally a patent umbilical vein.

A 6th group (Mongour), tributaries from the coronary ligament of the liver.

The following communications exist between the portal and superior vena caval systems:—

- (a) Gastro-oesophageal anastomoses.
- (b) Anastomoses between the peritoneal veins and those of the diaphragm, and through these with the intercostal veins, the internal mammary vein, and the azygos and hemi-azygos veins.
- (c) Anastomoses between the para-umbilical or patent umbilical veins and the superior epigastric, and through it with the internal mammary vein.

Communications between the portal and inferior vena caval systems:—

- (a) Venous plexus of Retzius:—

(1) Anastomoses with haemorrhoidal veins (large and numerous).

(2) Anastomoses with opposite parts of intestinal canal: duodenum, ascending and descending colon, and rectum. Here the peritoneal parietal intestinal veins ('die peritonealen Darmwand-venen') communicate with the veins of the parts not covered by peritoneum, i.e. with tributaries of the inferior vena cava.

(3) Anastomoses of veins of the descending colon with the left renal vein.

(4) Tributaries from the peritoneum communicate with each other, and drain both into the portal and inferior caval systems. These tributaries correspond with the 'Plexus of Turner.'

(b) Anastomoses between the peritoneal venous plexus in the posterior layer of the anterior abdominal wall and the veins of the visceral peritoneum on the one hand (portal system) and on the other with the subcutaneous veins of the anterior abdominal wall. The latter communications take place either through a patent umbilical vein (Baumgarten), or through one or more para-umbilical veins (Sappey): further, through perforating tributaries in the linea alba and recti muscles.

(c) Right and left renal-portal anastomoses.

(d) Finally abnormal anastomoses:—e.g. porto-spermatic, porto-uterine, porto-prostatic, etc.

The above anastomoses of the portal and other venous systems have been given in full as they apply with nearly equal force to the mesenteric.

SECTION VI

SYMPTOMATOLOGY

IN dealing with this branch of the subject we are beset at the outset with considerable difficulties owing to the diverse forms under which the disease appears. It may be said however that there are two main clinical types,—the acute, and the chronic. At the same time it must be admitted that the dividing line between the two classes is often far from clear, and that the distinction therefore is apt to become somewhat arbitrary.

For statistical purposes in this series the cases have been divided into those with a course of a week and under, and those lasting for 8 days and over. Under this classification it is found that the percentage of acute cases resulting from arterial lesions is very much higher than in those due to venous thrombosis. The actual figures are given below:—

	7 days and under	8 days and over
Arterial obstructions	85 cases—76·6 %	26 cases—23·4 %
Venous obstructions	54 cases—54 %	46 cases—46 %
Coexistent arterial and venous obstructions	14 cases—82·4 %	3 cases—17·6 %

The older view that arterial cases tended to be more acute than venous ones appears therefore to have some foundation. The high percentage of acute cases following a double vascular lesion is in accordance with what might be expected.

Jackson, Porter and Quinby found that out of 121 cases the percentage of those running a course of 7 days and under was almost exactly the same in venous and arterial occlusions: in the former it was 74 %, and in the latter 72 %.

Symptomatology

The following tables give the incidence according to age and sex :—

	Male	Female
Arterial obstructions	93 cases	76 cases
Venous obstructions	88 „	43 „
Coexistent arterial and venous obstructions	12 „	4 „

Total in which sex was stated—316 cases.

Age, years	Arterial obstructions	Venous obstructions	Coexistent arterial and venous obstructions
1—10	1 case	2 cases	1 case
11—20	9 cases	11 „	
21—30	19 „	28 „	2 cases
31—40	24 „	35 „	4 „
41—50	37 „	16 „	5 „
51—60	31 „	15 „	2 „
61—70	29 „	14 „	4 „
71—80	13 „	3 „	
81 and over	3 „		

Total in which age was stated—308 cases.

The youngest patient of the series was that of a child of five, but Lagane mentions one of one month. The oldest was a woman of 85.

It will be convenient to consider first of all the group of symptoms that are commonly present after a vascular occlusion in the mesentery, and then to discuss the manifestations of some of the prodromal conditions.

The symptoms of the disease are very much the same whether they are due to arterial or venous occlusion or both together. The onset is usually acute, though the conditions which led up to it may have been present for months or even years previously. Thus a patient who has a valvular lesion of his heart, or hepatic cirrhosis, or chronic Bright's disease, is suddenly seized while engaged in his ordinary occupations with severe abdominal pain. This is accompanied or soon followed by nausea, and possibly faintness, vomiting of blood-stained matter, and diarrhoea; the last often contains blood, but on the other hand the bowels are sometimes quite normal until the onset of constipation. The

patient is collapsed, the face covered by cold sweat, and the expression anxious. The pulse is rapid and small, and soon assumes the type found in cases of severe internal haemorrhage. The temperature is usually normal or subnormal, though there are numerous exceptions. The abdomen becomes distended and tender at an early stage, and on this account difficult to explore by palpation. After a few hours the pains become less intense, though the other symptoms continue unabated: constipation supervenes and may be absolute, and is associated with progressive distension of the abdomen. After a relatively 'latent' period of variable duration (a few hours or even days) there is a recrudescence of the pains which are occasionally aggravated by the occurrence of hiccough. Vomiting becomes almost continuous, the vomited matter being dark coloured and extremely offensive. Constipation continues, though there may be a terminal passage of blood by the bowel just before death. This stage of the disease may last for a few hours, or one or two days before death takes place. Towards the end the patient is often partially or completely comatose.

Nothnagel (quoted by Zesas) divided cases of infarction into two groups, viz., those in which diarrhoea and melaena preponderated, and those manifesting the symptoms and signs of acute intestinal obstruction; but Neutra expressed doubts as to whether any cases had occurred in which paralytic symptoms had not been preceded by those due to irritation. The latter authority attributed the supposed absence of initial symptoms preceding obstruction to inaccuracy in the history, or failure of memory on the part of the patient. Sprengel, as we have seen above, attempted to correlate the cases in which early intestinal obstruction had taken place with the presence of anaemic gangrene, and the cases shewing irritative symptoms with haemorrhagic infarction; but unfortunately for this view, early obstruction often supervenes in cases of haemorrhagic infarction. As regards the nine cases of anaemic infarct however, four¹ and possibly a fifth² developed early obstruction; while of the remainder one³ developed no

¹ Leclerc and Cotte, Neumann (revd. by the first named authors), Sprengel, case No. 1 (original).

² A fifth (possible) case was reported by Grawitz.

³ Meyer.

gastro-intestinal symptoms at all before death, another¹ passed clear watery stools two days after the onset of symptoms, a third² had diarrhoea and vomiting (without blood) and no obstruction, and the last (a doubtful case)³ passed a copious evacuation of blood just before death.

Some of the symptoms and signs may now be considered individually. Pain is one of the most constant symptoms, its absence having been recorded in only a few instances⁴. It is often described as 'agonising,' and not infrequently it remains unaffected by morphia. It is generally colicky or spasmodic in nature, lasting for five or ten minutes at a time and passing off for an equal period (Gallavardin), or it may be continuous. According to Jackson, Porter and Quinby it is generalised throughout the abdomen in 51 % of cases, but it is often described as being most intense about the umbilicus or in the epigastrium. The point of maximum intensity again may be situated in either hypochondrium, in the lower abdomen, or in one or other loin, though the latter situation is rare.

As regards the origin of abdominal pain, the balance of evidence at the present day seems to shew that it can only be produced through agency of the parietal peritoneum, and that the stimulus which most readily elicits it is traction. Traction may be exercised directly or indirectly on the parietal peritoneum in cases of infarction (1) by excessive peristalsis dragging upon the mesentery, or (2) by swelling of the latter. Borszéky, basing his opinion upon Kader's experimental observations, considers that the pains in infarction are due to spasmodic contractions of the intestine that has been deprived of oxygen by circulatory disturbances; but were this the only explanation the pains might be expected to cease within an hour or two of the onset⁵, i.e., directly the coil has become paralysed. It is much more probable however that the

¹ Niederstein.

² Zimmermann (revd. by Talke).

³ Roper.

⁴ Trübel, Drozda (revd. by Orthner), Aronsohn, Watson (both revd. by Deekart). The absence of pain in the first three of these cases was due to coma. (For other cases in which symptoms were absent, *vide* last page of this section.)

⁵ Active peristaltic movements were observed by Kader after severe strangulation for the first three or four minutes, and after slight strangulation (veins only being compressed) for a few seconds or at most a few minutes: then followed weakness of the intestinal muscle, and in a few hours absolute paralysis.

infarcted coil acts as a source of excessive peristalsis in the rest of the bowel, and thus produces abdominal colic in the usual way. This supposition is quite consistent with the observed facts regarding the extreme rarity of visible peristalsis in cases of infarction; for clinically, visible peristalsis is a phenomenon that is inconspicuous unless the bowel is hypertrophied and distended, and the abdominal wall relaxed, the first and last of which conditions are rare in infarction.

The other potent factor in bringing about traction on the parietal peritoneum—swelling of the mesentery—is often present in an extreme degree in cases of mesenteric circulatory obstruction, and probably accounts for the very great severity of the pain and shock.

Associated with pain there is generally extreme tenderness of the abdomen, though in 30 % of Jackson, Porter and Quinby's series of cases its absence was definitely recorded. These authors state that when present it was generalised in 67 %, localised in the epigastrium in 7 %, about the caecum in 7 %, and in the umbilical region in 8 %. It is probably most marked in the region overlying the coil most involved, but the evidence for this is uncertain. In those cases where a mass has been felt the latter has always been tender. According to Lagane there is sometimes great tenderness over the abdominal aorta in cases of arterial thrombosis associated with aortitis.

The following table comprises all the cases in the present series in which data are available regarding the presence of diarrhoea, melaena, and constipation:—

Cases of	Diarrhoea	Melaena	Constipation	Absolute Constipation	Constipation then diarrhoea	Diarrhoea then constipation
Arterial occlusion ...	51	60	36	13	3	8
Venous ditto ...	29	38	30	17	1	4
Arterial and venous occlusions together	7	5	6	1	0	3

From the figures given above, which are necessarily very incomplete, it appears that diarrhoea is slightly more frequent than constipation in these cases, but that the two together are not found so commonly as would be expected from the statement

made by Borszéky¹; this author regards neither diarrhoea nor constipation as a constant symptom, but attaches importance to the succession of constipation upon diarrhoea.

The absence of constipation is explicable in many cases by the death of the patient before the paralytic stage has been reached.

The statements made by Gallavardin² and Deckart³ to the effect that the stools are nearly always liquid appear to be rather too general, though as we have seen, diarrhoea is often present.

The stools may be merely blood-stained, or may contain large quantities of blood: in some cases evacuations of almost pure bright red blood have been reported, while in others the stools have been black and of tarry consistence. It is probable that in many cases where blood is present in only small quantities it is overlooked, particularly when it is not associated with diarrhoea. Sauvé expressed the opinion that blood in the stools was far more frequent in cases of arterial than of venous occlusion, but this view is not supported by the above figures, when the relative frequency of arterial and venous lesions is taken into account. Sprengel⁴ considers that the occurrence of bloody vomit or bloody diarrhoea by the passage upwards or downwards of sanious effusion through the sound intestine depends, so to speak, upon accidental conditions; that the quantity of the effused blood, the length and dilatability

¹ The following passage may be quoted from his work:—‘Als konstante Symptome der Erkrankung können wir also weder die blutige Stuhlentleerung, noch die Obstruktion betrachten, sondern beide zusammen in dem Sinne, dass Anfangs Diarrhöe vorhanden ist, öfters mit blutigen Stuhlentleerungen, später aber eine hartnäckige und jeder Behandlung trotzende Stuhlverhaltung auftritt’ (*loc. cit.* S. 172).

² Gallavardin (*loc. cit.* p. 958) says:—‘La diarrhée ne manque à peu près jamais, elle précède les hémorragies intestinales et en tient lieu en quelque sorte lorsque celles-ci font défaut.’

³ Deckart makes the following statement (*loc. cit.* S. 543):—‘Die Stühle sind meist diarrhoischer Natur, oft stark bluthaltig, ja manehmal aus fast reinem Blut bestehend, und zeichnen sich mitunter durch einen aashaften Geruch aus.’

⁴ Sprengel in his work (*loc. cit.*) expresses the following view:—‘Ob das in den gesunden Darm übertretende Blut nach oben oder unten gelangt und als blutiges Erbrechen oder blutiger Durchfall nach aussen in Erscheinung tritt, wird sozusagen von Zufälligkeiten abhängig sein. Die Menge des ergossenen Blutes, die Länge und Ausdehnungsfähigkeit des infarcierten Darmtheils, seine Lage im Abdomen, im späteren Verlauf Adhäsionen und dadurch erfolgende Abknickungen des Darms werden hier Wirkungen ausüben können, die sich selbstverständlich jeder Berechnung entziehen.’

of the infarcted segment of intestine, its position in the abdomen, and later on adhesions and kinks produced thereby, will be capable of producing effects that will obviously elude every calculation.

In most cases where diarrhoea obtained, the stools were very thin and watery, and very abundant. In some cases, particularly when blood was present, they were extremely offensive. Shreds of mucous membrane¹ have been observed in a few instances.

Defaecation is not uncommonly very painful, and associated with tenesmus.

Constipation in cases of infarction may be partial (faecal matter being retained, but flatus passed), or absolute (retention of both faecal matter and flatus), and is brought about by a number of factors of varying importance. After the initial stimulation by carbon dioxide poisoning (observed experimentally by Kader) the affected coil loses its power of contractility, a function which is further prevented by engorgement and infiltration of the walls with serum and blood. The coil thus affected offers a passive obstruction to the contents of the intestine above, though according to Gobiet, this obstruction is not insurmountable if the infarct is small. The mobility of the coil is seriously interfered with by gas accumulation, and coexisting oedema and haemorrhagic infiltration of the mesentery. If peritonitis supervenes, adhesions about the infarcted coil may lead to kinking of adjacent coils which have not yet become involved in the circulatory disturbance. In addition to the above factors, the absorption of toxic bodies resulting from stasis and decomposition in the intestinal contents may tend to bring about paralysis in the rest of the intestine.

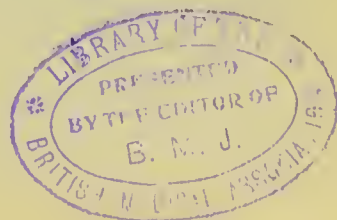
Constipation from the time of onset of symptoms is rare: Neutra was able to collect five cases² only, to which may be added two others³.

Jackson, Porter and Quinby state that towards the end of the paralytic stage of the disease blood is evacuated per rectum in 10 % of cases.

¹ Monro and Workman, Gull, Fagge, Claisse and Abrami, Späth (revd. by Neutra), Ott (revd. by Talke), Michaelis.

² Neutra reviews the following cases:—Virchow, Parensky, Cattani, Kaufmann, and Deckart.

³ Talke, original case No. 4.



Vomiting is one of the earliest and most constant symptoms, and it may persist until death. At the onset it is generally alimentary, but rapidly becomes bilious and then stercoraceous or sanious. No case of 'faecal' vomiting, in the strict sense of the word, is on record.

Though the vomited matter first consists of gastric contents, the latter may be mixed with a large amount of blood that has regurgitated, more especially if the vascular lesion is situated near the upper end of the jejunum.

Haematemesis is not nearly so common as melaena; out of the present series of cases it was reported in the following:—

Arterial occlusions	Venous occlusions	Coexistent arterial and venous occlusions
24 cases	25 cases	3 cases

When the vomit contains blood it may be bright red, or more commonly dark brown or black, and 'coffee-ground' in type. Much stress was laid upon the presence of blood by Gerhardt, Kussmaul, and Nothnagel (quoted by Roussel), on account of its supposed diagnostic significance, and it was even suggested that on this account the vomited matter should be examined microscopically.

Hiccough is a symptom which causes a great deal of distress to the patient, but which is rather a manifestation of the onset of peritonitis than that of a mesenteric vascular disturbance.

Abdominal distension is a sign that generally appears at an early stage (within a few hours of the onset), and increases as the disease advances. It is one of the most constant of the signs, as may be seen from the following figures. Out of 155 cases in which particulars are given as to its presence or absence we find it was present in:—66 cases of arterial occlusion; 59 cases of venous occlusion, and 10 cases of arterial and venous occlusions together.

The distension may be generalised, or localised over a small area. Sprengel believes that in the early stage of a mesenteric circulatory obstruction local distension indicates gas accumulation in the affected coil. This would account for the local percussion note, which is at first tympanitic: but as the intestinal walls become thickened by oedema and blood extravasation this note

may gradually become subtympantic and finally dull. At a later stage the note is generally tympanitic over most of the abdomen, with the above exception and with the exception of the flanks where it is often dull from the presence of free fluid in the peritoneal cavity. Free fluid is particularly likely to be present if there has been antecedent disease of the heart, or hepatic cirrhosis; it is also more likely to be found in cases complicated by peritonitis.

Adequate palpation of the abdomen is generally rendered impossible by the distension and tenderness, both of which may become extreme; examination is rendered difficult also by the reflex muscular rigidity that is present in many cases, all respiratory movements being confined to the thorax. Yet in spite of, or in the absence of these signs, a tumour or ill-defined mass has been felt in a few cases, while in others an ill-defined localised resistance has been elicited.

A tumour was felt in five cases of arterial and in six of venous occlusion. Out of the eleven cases the tumour was situated about the umbilicus in four¹, in the right iliac fossa in three² (one of which however was not confirmed)³, and in the lower half⁴ of the abdomen, in either flank⁵, and in the pelvis⁶ in one respectively. The tumour was generally rounded or sausage-shaped, rather doughy or hard in consistence, fairly well- or else ill-defined, slightly movable but unaffected by respiratory movements, and tender. In one of the above cases the tumour was felt only with the aid of an anaesthetic.

Localised areas of resistance were reported in four arterial cases, four venous, and in two where there were double vascular lesions. Out of these the resistance was localised about the umbilicus in three cases⁷, in the right hypochondrium⁸, the right lower abdomen⁹, the epigastrium¹⁰, the right¹¹ and left¹² iliac fossae

¹ Jackson, Porter and Quinby, No. 6, de Jong, No. 18, Polya, No. 2, Thévenot and Rey.

² Gordon, Munro, Rodger.

³ Rodger.

⁴ Gould.

⁵ Lecène (revd. by Sauvé), Thévenot and Rey.

⁶ London Hosp. P. M.'s 1908, No. 1144.

⁷ Brunner, Polya, No. 4, Sprengel.

⁸ Reitzenstein.

⁹ Jackson, Porter and Quinby, No. 4.

¹⁰ Packard (revd. by Jackson, Porter and Quinby).

¹¹ Borszéký.

¹² Case No. 3 (original).

in one case respectively: in the remaining two cases resistance was described in the right iliac¹ and right hypogastric² regions respectively, but in both cases the patient recovered without operation so that the cause of the resistance was not discovered.

The areas of resistance were in most cases tender, ill-defined, and gave the variable percussion note of an infarcted coil.

Visible peristalsis is extremely rare in cases of mesenteric circulatory obstruction. Out of about 360 cases in this series it was reported in 4 only³.

The temperature at the onset may be normal, subnormal or raised. Jackson, Porter and Quinby observe that a subnormal temperature is frequent. To quote these authors:—‘The temperature usually falls below normal, though not infrequently an increase is observed, which is perhaps due to accompanying processes, such as peritonitis, endocarditis or toxic absorption from the intestinal contents.’ Zesas⁴ and Sauvé remark that a subnormal temperature is observed in nearly all cases, a generalisation with which Deckart does not agree, having failed to observe it in any one of his own cases or to collect more than eleven instances from the literature.

It is possible that subnormal temperatures would be reported more often if the cases came under observation at an early stage of the disease; in actual fact, the temperature has often risen before accurate records can be taken. This is a consideration that has to be borne in mind when we come to examine the statistics: nevertheless there is scarcely sufficient evidence for the assertion that the temperature at the onset is nearly always subnormal. Out of 112 cases regarding which details are available the temperatures were:—subnormal in 22, subnormal to normal in 2, normal in 15, normal to pyrexial in 1, subnormal to pyrexial in 6, and pyrexial in 66.

The pulse is commonly that of a severe internal haemorrhage, small, hard, rapid (100 to 140 or 150) and in many cases irregular.

¹ Bayer (revd. by Talke).

² Aufrecht.

³ Deckart, de Jong, No. 8, Ballance, Leech.

⁴ Zesas quotes Cohn's explanation for a subnormal temperature in these cases. According to this observer it is due probably to loss of blood, associated with an effort on the part of the heart to overcome the increased peripheral resistance in the mesentery.

Of course in cases where the vascular lesion is not extensive, the pulse may not shew much change beyond a slight acceleration.

The respirations are accelerated soon after the onset in severe cases (i.e., in those which do not terminate in spontaneous recovery), and may be entirely thoracic, the abdomen being held rigid by reflex muscular spasm. The depth of each inspiration is correspondingly diminished, a condition that may give rise to a variable degree of cyanosis. Oedema of the bases of the lungs may result from immobility of the diaphragm, combined in some cases with an antecedent cardiac lesion.

Coma supervenes during the last few hours of life in some cases. It generally comes on gradually with progressive loss of strength, and the patient may lose consciousness completely several hours before death. Coma is probably due to acute toxæmia from the intestinal tract, associated with exhaustion following prolonged very severe pain.

OTHER CLINICAL TYPES OF MESENTERIC VASCULAR OCCLUSION.

As we have seen in an earlier part of this paper, only a certain proportion of vascular occlusions lead to infarction, and it is rare even for cases of infarction to present more than a few of the classical signs and symptoms described above. In other cases the terminal clinical picture may be that of the most acute form, though the mode of onset has been more gradual. In fact, it may be said that even in those cases where gastro-intestinal symptoms and signs suggesting disease in the mesenteric vessels have been present for a long time—months or perhaps years—the end of the disease may run, so to speak, an acute course.

Although in any given case the clinical manifestations are rarely sufficiently clear to justify a certain diagnosis of venous as against arterial obstruction, or *vice versa*, yet it may not be unprofitable to consider the previous clinical histories of different morbid-anatomical types.

In general it may be said that a very large proportion of cases of mesenteric arterial occlusion, particularly of embolism, present

histories of cardio-vascular disturbances¹. Thus, the patient has complained of shortness of breath, palpitation and swelling of the feet perhaps for years; another has been the subject of extreme anasarca with enlargement of the liver, ascites, and other signs of failure in compensation of a dilated heart; another has had one or more attacks of rheumatic fever associated with endocarditis, which may or may not have led to subsequent inconvenience. Other cases again have had definite embolic attacks in different parts of the body, giving rise to hemiplegia, aphasia, gangrene of a limb from obstruction of the main artery, haemoptysis from small infarctions of the lung, or haematuria.

In another type of case the patient has suffered from chronic nephritis and arterio-sclerosis², with or without cardiac hypertrophy. Lagane remarks that the estimation of the blood-pressure from the radial arteries in these cases is no gauge of the pressure in other parts of the arterial system. He says that the tension indicated may be raised, normal or even subnormal in cases of arterial atheroma. Patients suffering from atheroma of the aorta are, in his opinion, particularly prone to intestinal troubles, owing partly to this condition, and partly to the frequently associated mesenteric arterial disease.

It is not uncommon for patients who finally succumb to the effects of mesenteric arterial occlusion to have had gastro-intestinal disturbances³ of a more or less chronic character. Flatulent distension, 'heart-burn,' attacks of nausea and vomiting with or without relation to food, diarrhoea, constipation, and other similar troubles are frequently reported, but it is difficult to say how often the symptoms are referable to prodromal arterial disease, and how often they are merely fortuitous.

From the point of view of arterial thrombosis the most important antecedent conditions are probably atheroma of the mesenteric arteries, and arterio-sclerosis and endarteritis of the parietal branches in the intestine. Sclerosis in the mesenteric

¹ Cardio-vascular disturbances were reported in the histories of 86 cases of mesenteric arterial occlusion in the present series.

² Lagane's case No. 3, and Jackson, Porter and Quinby's cases Nos. 9 and 23 may be mentioned as examples.

³ There was a history of past gastro-intestinal disturbances in 28 cases of mesenteric arterial occlusion.

arteries and their branches is responsible for a clinical picture that has received the names, 'Dyspragia intermittens angiosclerotica intestinalis' (Orthner), 'Intermittierende anämische (or ischämische) Dysperistaltik' (Schnitzler), 'Crises de méiopragie intestinale' (Charcot¹, Josué², Sauvé, Lagane, and other French authors). It may conveniently be called 'Intermittent mesenteric claudication' on account of its aetiological similarity to the condition ('Intermittent claudication') in horses, described first by Charcot (quoted by Orthner).

Clinically, atheroma, arterio-sclerosis and endarteritis in the mesenteric arteries and their branches lead to transitory symptoms that are in many cases periodic. The patient complains of colicky abdominal pains associated with distension which may be generalised, or localised in one part of the abdomen. Constipation is common, though there may be copious evacuations of watery mucous stools that are occasionally streaked with blood. Nausea, eructation and vomiting are frequent symptoms. In severe cases respiration may be impaired on account of the abdominal distension, and the patient shews signs of extreme collapse. Orthner remarks that the crampy pains and feeling of distension are most commonly situated about the umbilicus, and that they begin, in the early stages of the disease, 1 to 1½ hours after food and last for about 2 hours. Though the attacks at first bear some relation to food they are in the later stages often brought on by other stimuli, such as exertion, mental effort, emotion, or the assumption of the horizontal position. Between the attacks the patient feels quite well as a rule, but a certain amount of abdominal discomfort may persist. The condition is essentially a chronic one, varying from six months³ to five years⁴ in duration, the attacks recurring every day, or at long intervals⁵.

The pain and local meteorism are probably due to temporary spasm of the arteries of supply, and paralysis of corresponding

¹ Quoted by Orthner.

² Quoted by Lagane.

³ Markwald's case (quoted by Orthner), not included in this series.

⁴ Schnitzler (four years duration). Lépine (five years).

⁵ Zesas remarks that von Engelhardt has for a long time pointed out the clinical similarity between 'intestinal neurosis' and the phenomena of mesenteric arterio-sclerosis, and has said that during these neuroses, vaso-motor disturbances are observed in other parts of the body, strongly suggesting a disturbance in the circulation.

segments of intestine. Both Orthner and Lagane, who have made a special study of the disease, insist upon the invariable absence of visible peristalsis.

Intermittent mesenteric claudication may or may not prove fatal: on the one hand thrombosis of the affected vessels may supervene after a variable period of rigidity of their walls; while on the other, death may result from an intercurrent affection. In Schnitzler's case (already referred to in connection with collateral compensation) old standing thrombosis was found associated with recent intestinal infarction. In those cases that lead to thrombosis, the symptoms of intermittent mesenteric claudication generally become more intense, and finally to these symptoms are superadded infarction as described above.

Sauvé has described a variety of intermittent mesenteric claudication in which a very severe attack precedes the terminal manifestations by about two or three weeks (though the length of the interval is not emphasised). After the severe attack referred to there is a complete remission of the symptoms till the onset of the fatal attack. To this clinical type Sauvé has applied the term '*début en deux temps*,' and quotes as an illustration the case reported by Auvray: in this case the latent period between the two attacks was about 10 days. One of Lagane's cases (No. 3) is another instance of this type: the patient was a man of 53 who had had syphilis and gonorrhoea, was very alcoholic, and subject to renal colic; during the last three years he had had manifestations suggesting mesenteric arterio-sclerosis: he was admitted for acute abdominal pain, distension, melaena and haematemesis; 'perforated gastric ulcer' was diagnosed and operation advised; this was refused, and the patient left hospital a week later. Three weeks later (i.e., a month after the first acute attack) he was admitted moribund. At the autopsy infarction of the intestine was found, with extreme sclerosis and endarteritis of the parietal branches of the superior mesenteric artery in the intestine.

The class of patient most commonly affected by atheroma of the mesenteric arteries and arterio-sclerosis of the branches in the walls of the intestine has been described by Lagane, from whose work the following quotation may be given¹:—

¹ Lagane, *Presse Médicale*, Dec. 1911, p. 1025.

‘Le premier (i.e. atheroma) semble exister le plus souvent avec des lésions chroniques de l’aorte et du reste du système vasculaire, se rencontre souvent chez des syphilitiques, ordinairement chez des gens ayant passés la cinquantaine et athéromateux ; la seconde (i.e. parietal arterio-sclerosis) relève plus souvent d’affections aiguës ou chroniques de l’intestin et se voit chez des sujets plus jeunes, alcooliques, saturnins, syphilitiques souvent, chez d’anciens typhiques ou des malades anciennement atteints d’infections aiguës ou chroniques de l’intestin, enfin chez des malades dont l’intestin a eu déjà, pour des raisons quelconques, à subir des lésions curables d’infarcissement.’

As we have seen, a certain number of cases of mesenteric embolism are on record which were associated with purpuric manifestations, and for this reason they may be described as a separate clinical class.

In most of the cases the patient was less than 50 years of age, and was suffering from either a recent or a chronic heart lesion, generally of rheumatic origin : in four cases¹ and possibly a fifth² however the cardiac lesion was septic (pyaemic) in origin. The purpuric eruptions varied from a few spots on the hands up to extensive cutaneous haemorrhages covering a large part of the body. The commonest sites of the rash appear to be the precordium and limbs where it may be localised about the joints.

In some cases the eruption was associated with pains and fluid in the joints, which became swollen and tender³.

Abdominal symptoms of greater⁴ or minor severity⁵ may accompany, or follow the eruptions, or there may be several crops of purpuric haemorrhages : again the abdominal symptoms sometimes precede the cutaneous haemorrhages. The gastrointestinal symptoms vary in intensity according to the extent of the embolic process. Thus in Talke’s case the first embolic attack that affected the intestine did not prove fatal, the clots merely

¹ Booth, London Hosp. P. M.’s 1908, No. 1153, 1910, No. 500, and 1911, No. 50.

² Possibly the case reported by Moos.

³ Talke, Wagner (revd. by Talke).

⁴ London Hosp. P. M.’s 1910, No. 500, Talke, Moos.

⁵ Gimard (revd. by Talke), Wittmann. In Jackson, Porter and Quinby’s case No. 25 there was a complete absence of abdominal symptoms, although infarction occurred : the age of the patient was 44.

closing a number of minute vessels: it was only after the closure of two of the larger branches of the superior mesenteric that death took place. The symptoms usually consist of acute abdominal pains—which are independent of food—, nausea, vomiting and diarrhoea. The abdomen is distended, and there may be blood in the vomited matter, or in the stools, or in both. If the mesenteric embolism leads to ulceration of the intestine, portions of mucous membrane may be evacuated in the stools.

The duration of the disease depends mainly upon the gravity of the intestinal lesion, though in some cases, particularly in those due to pyaemia, the associated conditions may be determining factors.

In Talke's case the abdominal pains, slight at first, started four weeks before the patient was first seen, and death did not take place for another fortnight. This is probably an unusually long history, the intestinal and cutaneous symptoms and signs occurring generally only a few days before death.

Mesenteric venous thrombosis may, as we have seen above, be caused by chronic changes in the liver, such as cirrhosis and syphilitic perihepatitis, not to speak of other conditions producing obstruction in the portal circulation. A type of case may therefore be described in which symptoms of mesenteric venous thrombosis are preceded by those of portal obstruction recurring at intervals for a variable length of time.

The manifestations of portal obstruction sometimes extend over a period of years¹, during which the patient is seized with attacks of severe abdominal pain, melaena, haematemesis, and epistaxis. The amount of blood lost is sometimes considerable and may lead to fainting attacks. Long intervals may elapse between the attacks of bleeding: in Spiegelberg's case the haematemesis and melaena continued for eight days, after which there was an interval of four months before the next attack. In the case reported by Alexander² the attacks of haematemesis and melaena took place about every two years during a period of six years.

The patient generally complains of digestive disturbances, accompanied by anorexia and nausea. The stools are often loose

¹ Two St Bartholomew's Hospital cases.

² Alexander (revd. by Neutra).

and of watery consistence, though the evacuations are painless. In Seitz's case¹ the motions were of this description, and were passed two or three times a day for four years.

Abdominal distension and ascites are common signs of this disease, and if the portal vein is occluded the spleen is generally enlarged. The veins of the abdominal wall are often dilated.

The terminal attack resembles the previous ones, save that it is much more severe, and gives rise to severe collapse: to the effects of portal obstruction are superadded those resulting from the downward spread of the clot into the mesenteric veins, with consequent infarction of part or the whole of the intestine below the duodenum.

It will be convenient to consider next a clinical type of venous thrombosis which originates from infective processes either in the pelvis or the appendix. Perhaps the most striking features of these cases are the length of the latent period in which the symptoms are entirely or nearly absent, and the acuteness of the disease when the symptoms have once developed. A further point of interest is that the blood-count in these cases often shews a marked leucocytosis, as might be expected in view of their septic origin.

In the cases resulting from sepsis in the female pelvic organs the latent period between the time of the causal infection and the onset of acute abdominal symptoms may vary from a few days up to six or eight weeks. During this time the patient feels either quite well or else has only trifling symptoms, as for example malaise or slight headache; but no vomiting, diarrhoea, constipation, or distension. Then suddenly acute symptoms supervene, accompanied perhaps by rigors, fever, and slight jaundice.

In Kölbing's case there were no symptoms of any description for the first 23 days after perfectly normal labour. In Fagge's case mesenteric thrombosis was preceded by phlegmasia alba dolens which began 16 days after 'protracted normal labour.' Broussin² reported a case in which the lochia became offensive on the second day after normal labour: there were associated phlebitis of the upper and lower limbs, and rigors: on the fourth day acute abdominal pains, vomiting and blood-stained diarrhoea supervened.

¹ Seitz (revd. by Borrmann).

² Broussin (revd. by Maucclair and Jacoulet).

Garmsen described a case in which the lochia became offensive 11 days after the birth of a dead sixth month foetus: the placenta was removed manually. The patient developed severe abdominal pains, diarrhoea and vomiting six weeks later.

In cases following appendicitis the latent period is no less striking, but the absence of symptoms is not so complete as in the cases following sepsis in the pelvic organs. The usual course of the disease may be briefly outlined as follows:—The patient is seized with an attack of pain in the abdomen, which may or may not be localised in the appendix region: vomiting follows, accompanied by diarrhoea or constipation. In a few days the symptoms subside and the patient feels able to get up. For the next 7 or 14 days there may be variable degrees of pyrexia, but there are no very marked symptoms. At the end of this latent period he is seized with acute pains in the abdomen, accompanied by rigors, fever, nausea, vomiting, and occasionally slight jaundice. The bowels are generally constipated, and there is often a leucocytosis. The rest of the course of the disease is usually that of the acute type described above, and unless operative measures are undertaken at once pylephlebitis or extensive mesenteric venous thrombosis supervenes.

In case No. 5 (original) of this series there was diminution of pain after the onset of the appendicitis. The appendix was removed on the 9th day (i.e. on admission to hospital), and though signs of venous thrombosis had not yet developed there were definite changes present already in the lower end of the mesentery: the thickening in the latter was taken at the time to be a mass of enlarged glands. On the 3rd day after removal of the appendix—which was purulent—the patient had a rigor, and the temperature rose to 103·8: constipation accompanied by an irregular temperature followed for the next two days, towards the end of which he vomited. On the evening of the 5th day after appendectomy, i.e. the 13th after the onset of the first symptoms, a second operation was undertaken, and the lower end of the ileum found to be bluish and congested, but viable.

In the other original case (No. 3) indefinite symptoms of thrombosis began to appear 10 days after the first symptoms of appendicitis. In this case there was an interval of about a week

during which all the symptoms were slight, and the patient felt comparatively well. The clinical picture of the terminal disease was not so rapid in its development as in some of the cases: pain and constipation started on the 11th day but there was no vomiting till the 16th, and there was never any rigor. Infarction of part of the small intestine was found 17 days after the onset.

A certain number of cases of mesenteric vascular occlusion have been attributed to operative procedures. The age of the patients affected ranged from 26 to 75, and the operations that had been performed were the following:—For strangulated hernia (Adénot)¹, strangulated hernia reduced by taxis (Patal)², gastro-enterostomy for ulcer of the stomach, and for pyloric stenosis (Gobiet and Maylard respectively), radical cure of inguinal hernia with omental adhesions (Ullmann)³, removal of myxosarcoma of 14·5 kilos weight from abdomen (Braquehaye)⁴, myomectomy (Hegar), myomectomy with removal of part of the transverse colon (Adénot)², amputation of the leg for injury, followed by sepsis (Faber), excision of the elbow (Ponfick)⁵, and finally hemithyroidectomy for Graves' disease (Maylard). One case which does not belong strictly to this group followed 'ptomaine poisoning' (Thomas).

In some of these cases the abdominal symptoms developed within a few hours after operation⁶, or else death took place almost at once⁷; while in others there was a latent period of a week⁸ or fortnight⁹ without symptoms.

The symptoms usually consisted of acute abdominal pains, diarrhoea or absolute constipation, melaena, vomiting and abdominal distension.

In five cases¹⁰ the arteries were involved, in four¹¹ the veins,

¹ Adénot (revd. by Neutra).

² Patal (revd. by Sauvé).

³ Ullmann (revd. by Maucclair and Jacoulet).

⁴ Braquehaye (revd. by Maucclair and Jacoulet).

⁵ Ponfick (revd. by Deckart).

⁶ Maylard (thyroidectomy).

⁷ Adénot (revd. by Neutra).

⁸ Ullmann (revd. by Maucclair and Jacoulet), Gobiet.

⁹ Ponfick (revd. by Deckart).

¹⁰ Adénot (two cases, *ibid.*), Faber, Hegar, Ponfick (*ibid.*).

¹¹ Maylard (two cases), Thomas, Gobiet.

in one¹ both artery and vein, and in two² it was doubtful whether the lesion was in the artery or in the vein.

The last clinical group to be considered is that in which abdominal symptoms were entirely absent. Out of this group, two cases³ followed labour in patients with endocarditis, four patients⁴ had diseases of the lungs or pleura, one⁵ died from an attack of apoplexy, two⁶ from chronic nephritis, one⁷ from arterio-sclerosis with ascites, one⁸ had gangrene of the leg and malignant endocarditis, one⁹ suddenly developed weakness of the legs associated with hemianopia, and finally one¹⁰ gave signs merely of senile cachexia and arterio-sclerosis.

Only two of these patients were 70 years of age and over, and one was 66: all the others were under 60, and the youngest was 15. The only patient who can be said to have had mental symptoms was the one who died from apoplexy: all the rest appear to have retained consciousness till shortly before death. Maclaure and Jacoulet express the opinion that pain in this disease is slight in the aged, in lunatics and in the cachectic; but from about 360 cases in this series we have been unable to obtain evidence in support of this view.

¹ Patal (revd. by Sauvé).

² Ullmann (*ibid.*), Braquehay (revd. by Maclaure and Jacoulet).

³ Allen, London Hosp. P. M.'s 1911, No. 50.

⁴ Altmann, Aronsohn (revd. by Talke), London Hosp. P. M.'s 1907, No. 1133, 1908, No. 1153.

⁵ Ginsburg (revd. by Jackson, Porter and Quinby).

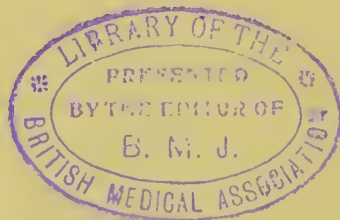
⁶ Watson; Jackson, Porter and Quinby, No. 23.

⁷ Körte (revd. by Kölbing).

⁸ London Hosp. P. M.'s 1910, No. 123.

⁹ Jackson, Porter and Quinby, No. 25.

¹⁰ Meyer.



SECTION VII

DIAGNOSIS

Occlusions in the mesenteric vessels give rise to lesions that are extremely difficult to recognise. This may be realised from the fact that a diagnosis from the clinical manifestations was made in only 13 cases¹. Two circumstances have been responsible for the failure in recognition of the disease; in the first place the clinical picture is extremely variable, and in most cases very incomplete; and in the second place the condition has been regarded as so uncommon that it has frequently not even been considered. Mesenteric vascular occlusion is a disease that has now been met with sufficiently often to merit a place among possible alternative conditions when the diagnosis of an acute abdominal lesion is under consideration. This applies particularly to cases where there are present acute colicky abdominal pains, profuse bloody diarrhoea, or constipation, and intractable vomiting, associated with abdominal distension and grave constitutional disturbances.

¹ Claisse and Abrami ('Mesenteric embolism.' Embolism of the sup. mes. art.), Gerster ('Acute appendicitis; probably mesenteric thrombosis.' Thrombosis of tributaries of sup. mes. vn.), Mitchell (Jackson, Porter and Quinby's case No. 7, 'Mesenteric thrombosis.' Thrombosis of sup. mes. art.), Leclerc and Beutter ('Embolism of sup. mes. art.' Thrombosis probably secondary to embolism), Mollard and Monod ('Peritonitis from perforation, or mesenteric embolism or thrombosis.' Thrombosis of sup. mes. art.), Munro ('? embolism, ? malignant tumour.' Three infarcts but no embolus found), Piery and Dumas ('Mesenteric embolism.' Embolism of sup. mes. art.), Reitter ('Embolism of sup. mes. art.' Diag. confirmed), Reitzenstein ('Thrombosis of mes. vn., and paralytic ileus.' Diag. confirmed), Trübel ('Thrombosis of sup. mes. art.' Diag. confirmed), Thévenot and Rey ('Obstruction from ? cancer, ? volvulus, ? infarct.' Infarction from arterial thrombosis), Gobiet ('Thrombosis of the mes. vessels.' Thrombosis of sup. mes. vn.), Kanis ('Peritonitis, possibly portal or mesenteric thrombosis.' Portal and mesenteric thrombosis).

The symptoms and physical signs produced by obstructions in the arteries resemble so closely those due to venous thrombosis that it is generally impossible to distinguish clinically between the two lesions. Nevertheless, Gerhardt and Kussmaul (quoted by Zesas) laid down certain postulates for diagnosis in cases of mesenteric embolism, but it is probable that they are never all fulfilled in any given case. The postulates may be enumerated as they form a valuable guide to diagnosis:—

- (1) A source for the embolus (e.g. endocarditis).
- (2) Copious melaena, not to be accounted for by any portal obstruction (e.g. hepatic cirrhosis), or lesion of the intestinal mucous membrane.
- (3) Rapid and excessive fall of temperature.
- (4) Very severe colicky abdominal pains.
- (5) Later, distension of the abdomen, with tympanites and shifting dulness.
- (6) Earlier or simultaneous embolic phenomena in other parts of the body.
- (7) Often large haematomata between the layers of the mesentery, felt through the abdominal wall.

Schrötter (quoted by Neutra) says that if a clinical picture suggesting a mesenteric vascular occlusion occurs in a young individual with a heart lesion, or in an old patient with arterio-sclerosis, one should think of mesenteric embolism. Schrötter's opinion as regards the first class of case is probably correct, but one would expect a patient with arterio-sclerosis to develop thrombosis rather than embolism, more especially if he had been subject to attacks of intermittent mesenteric claudication, or abdominal crises associated with angina pectoris, or chronic nephritis.

Although the clinical manifestations of intestinal infarction are obscure in the vast majority of cases, it is probably less difficult to recognise the condition when it is due to embolism than when it is caused by any other lesion. The presence of endocarditis and embolic manifestations elsewhere gives a valuable clue to the nature of the abdominal affection.

With regard to venous thrombosis Gallavardin says that in the face of acute abdominal symptoms simulating peritonitis or intestinal obstruction one may make a diagnosis only if (1) there exist cirrhosis (atrophic) of the liver, or signs of pylephlebitis, or phlebitis in a vein elsewhere; (2) there occur blood in the stools or in the vomited matter.

DIFFERENTIAL DIAGNOSIS.

In discussing the differential diagnosis of embolism or thrombosis in the mesenteric vessels one is at once confronted with the difficulty of placing a limit to the number of conditions to be considered, as there are innumerable diseases with which it may be confounded. It would be necessary to write a text-book on the diagnosis of acute abdominal complaints in order to deal adequately with the numerous mistakes that have been made concerning this disease. Only those acute abdominal conditions will be dealt with therefore which bear the closest clinical resemblance to mesenteric embolism or thrombosis.

A condition that is particularly liable to be confused with infarction of the intestine is *intussusception*, more especially when the infarcted coil can be felt through the abdominal wall; there is absolutely no pathognomonic sign whereby intussusception can be excluded. It has been suggested that the variability in the consistence of the tumour on palpation is a point in favour of the latter, but the abdomen is generally too distended or tender in infarction for this sign to be of much value. Although in many cases it is impossible to make a differential diagnosis, yet the following points would be of value as a guide:—(1) The age of the patient. Infarction though it may occur at any age is more prone to affect adults between the ages of 30 and 70; acute intussusception on the other hand is a disease of childhood, though it may affect adults. (2) A history of one or more previous attacks characterised by marked tenesmus, and the passage of blood-stained mucus by the bowel would be in favour of acute intussusception; in infarction the amount of blood and faecal matter should exceed that passed in acute intussusception, except in those very rare cases where obstruction occurs directly after the onset, while the

passage of mucus (as distinguished from serous stools) is not common in infarction: tenesmus again is not so constant or prominent a symptom of infarction as of the other disease. (3) Complete remission of the symptoms between the attacks is a characteristic of acute intussusception which is not shared by infarction; in the latter disease, after the onset it is most exceptional for there to be a period of complete freedom from symptoms, though the pain often becomes less severe for a time; the distinction from acute intussusception may become impossible however in cases of infarction that have been preceded by one or more severe attacks of 'intermittent mesenteric claudication.' (4) If on abdominal examination a sausage-shaped mass is felt, one end of which is palpable per rectum, intussusception is more probable than infarction.

To sum up these points of difference: mesenteric vascular occlusion has been reported in 1·3 % of cases in patients in the first decade of life, and an abdominal tumour has been felt in 5·7 % of cases of infarction: initial intestinal obstruction was observed in 2 % of all cases of mesenteric vascular occlusion of which data are available, and mucous stools are not common whether the vascular lesion has or has not led to infarction.

The clinical picture of chronic intussusception is not nearly so grave as that of a mesenteric vascular obstruction, and on this account the diagnosis will not be considered.

The next condition to be considered is *volvulus*. Though the symptoms and signs may be the same in volvulus as in infarction, yet there are certain manifestations of the former disease that differ from those which are more often met with in the latter. Absolute constipation associated with early extreme abdominal distension are signs that would favour a diagnosis of volvulus; for as we have seen it is extremely rare¹ for absolute constipation to occur at the onset in cases of infarction, and though distension appears early it does not reach the same degree as it usually does in volvulus till the later stages of the disease. A history of one or more previous attacks would favour a diagnosis of volvulus, but it is important to remember that cases of chronic portal obstruction

¹ Talke (absolute constipation at onset), Deckart (absolute constipation a few hours after onset, at which time there was diarrhoea).

leading as a terminal event to descending mesenteric venous thrombosis with infarction may present histories of melaena, haematemesis, acute abdominal pains and perhaps absolute constipation.

Neutra remarks that in volvulus of the sigmoid it is impossible to introduce more than a small amount of fluid per rectum, whereas in infarction there is no such limitation.

Stenosing carcinoma of the colon may present a clinical picture indistinguishable from infarction, but in the former disease there is generally a more definite history of gastro-intestinal disturbances associated with wasting. On the other hand, cases of arterial thrombosis due to arterio-sclerosis, and of venous thrombosis secondary to portal obstruction may give histories of recurring or chronic abdominal symptoms. A point which may be of value in the diagnosis is the absence of visible peristalsis in infarction¹, and its occurrence in chronic intestinal obstruction from any cause.

The symptoms and signs of internal strangulation are practically the same as those of infarction. It is only by taking into account coexisting morbid conditions, as for example a valvular cardiac lesion, an infective process in the pelvic organs or appendix region, a chronic arterio-sclerosis, a chronic alcoholic cirrhosis of the liver or a syphilitic perihepatitis, that any clue can be obtained as to the nature of the case.

Zesas remarks that in internal strangulation the evacuations per anum end suddenly, and that haematemesis is extremely rare. He considers for these reasons that there should never be any confusion in the diagnosis. If the manifestations of intestinal infarction were more constant it might be possible to accept this view; but as it is we know that not only may haematemesis be entirely absent, but also that absolute obstruction may supervene quite suddenly in this disease.

Borszéký is of the opinion that a differential diagnosis is in most cases impossible.

The differential diagnosis of pathological *complications* which may follow an attack of *appendicitis* and give rise to *acute intestinal obstruction* is extremely difficult. The acute obstruction

¹ Visible peristalsis was observed in about 1·1 % of cases.

in such cases is the result of toxaemia, peritonitis, adhesions, or infarction of the intestine from venous thrombosis. In the case of the first two complications, the symptoms usually merge into those due to the primary lesion, whereas adhesions or infarction manifest their presence as a rule after an interval of variable duration. The temperature in peritonitis is more often raised than in infarction, though if the peritonitis is generalised the temperature is often normal or subnormal as in infarction. The pulse is rapid and often irregular in both, but in infarction it tends to be 'wiry' on account of the loss of blood¹. The pain and constitutional symptoms associated with kinking of the intestine from adhesions are generally much less severe than those produced by infarction.

The clinical picture presented by the sudden extension of an abscess arising from a gangrenous appendix sometimes resembles very closely that of mesenteric venous thrombosis originating from the same cause. Thus a patient may be admitted to hospital with the following history:—Sudden severe pain in the right iliac fossa, and other symptoms of appendicitis 14 days before; disappearance of the symptoms in 4 or 5 days; reappearance of acute pain 7 or 8 days later, with fever, constipation, vomiting, distension, rapid pulse, followed by rapid onset of collapse. With such a history, and in the face of ambiguous physical signs, it is quite impossible to differentiate between infarction on the one hand and peritonitis or abscess resulting from a gangrenous appendix on the other. If the vomit is dark coloured or contains bright red blood this will be a point in favour of infarction.

A case with a clinical history somewhat of this description was admitted to University College Hospital early in 1912, and a tentative diagnosis made of mesenteric venous thrombosis following appendicitis. The patient who was extremely collapsed died immediately after admission. The autopsy shewed the presence of a gangrenous appendix and a large abscess, but no trace of venous thrombosis.

The differential diagnosis between infarction and *acute pancreatitis* is one of great difficulty. In many cases of the latter

¹ Too much stress must not be laid upon this sign however as it is often extremely difficult to draw a distinction.

disease the course is apyrexial; the pain and shock are very severe, the pulse is small, rapid and often irregular, and vomiting is a prominent symptom; the small and large intestines become distended and their movements inhibited, with the result that almost complete obstruction follows; finally, a tender swelling can sometimes be felt in the epigastrium.

Gobiet reports a case where the diagnosis lay between acute pancreatitis and intestinal infarction. The patient was a man of 62: there was extreme collapse associated with tenderness of the whole abdomen, and a small scarcely countable pulse; the pains were agonising ('rasenden Schmerzen'), and complete obstruction was present, with vomiting; there was marked sclerosis of the arteries. On the day after admission the patient felt better, and a high enema brought away much faecal matter free from blood; the temperature rose to 39, and the pain became localised in the epigastrium. Laparotomy confirmed the diagnosis (made on the second day) of acute pancreatitis. The diagnosis was made in this case upon the absence of blood in the stool resulting from the enema: all the other symptoms and signs would have been equally consistent with infarction.

Among other possible diseases to be thought of while making a differential diagnosis are lead colic, and typhoid fever with perforation (Lagane).

The distinction between mesenteric venous thrombosis and a bad attack of haemorrhage in *hepatic cirrhosis* may be difficult, but in the former the manifestations are much more intense and the general state of the patient much more collapsed.

The diagnosis between a *superior* and an *inferior mesenteric lesion* need not detain us long, as it is a refinement that is not likely to be attempted in a disease that is itself difficult to recognise. Lumbar pains were supposed by Kussmaul (quoted by Zesas) to indicate obstruction of a vessel of the inferior mesenteric system, and peri-umbilical pains one of the superior artery or vein; more recently Lerat and Cleret have laid stress upon the same point. Litten said that tenesmus was a sign of an inferior rather than a superior mesenteric vascular occlusion, and added that tarry stools occurred in the latter and bright red motions in the former: with regard to the latter point we have already

quoted Sprengel's opinion¹ which appears to be a satisfactory explanation. Glycosuria was found experimentally after ligature of the superior artery, but not after tying the inferior (Kolisch, quoted by Deckart). All these signs however are of doubtful value.

The diagnosis of 'intermittent mesenteric claudication' does not strictly speaking come within the scope of this paper, but it must be briefly mentioned on account of the relation of this disease (as a predisposing factor) to mesenteric arterial thrombosis. When considering a history of recurring attacks of abdominal pain, flatulence, distension, meteorism of the stomach or intestine, and constipation or diarrhoea, one should bear in mind the possibility of intermittent mesenteric claudication when the patient presents one of the two following causes that predispose to it:— (1) Chronic nephritis in the old, and (2) syphilis in the young (Orthner). The symptoms of this disease are always more severe than the abdominal symptoms sometimes associated with angina pectoris; but it is to be remembered that the two diseases may coexist.

A valuable aid in the diagnosis of intermittent mesenteric claudication from chronic intestinal obstruction is the invariable absence of visible peristalsis. Akin is of the opinion that an examination of the vascular system will generally decide the diagnosis, signs of arterio-sclerosis being in favour of the former disease. If however one were to attach much importance to evidence of this nature, the diagnosis of intermittent mesenteric claudication might be made in almost any abdominal complaint affecting a middle-aged or syphilitic patient.

¹ *Vide* 'Symptomatology.'

SECTION VIII

TREATMENT

THE treatment of mesenteric vascular occlusion up to the present has been far from satisfactory. In certain of the milder cases devoid of paralytic symptoms efforts have been made to treat haemorrhage, diarrhoea, vomiting, etc., by medical means. How many cases of mesenteric vascular obliteration have been successfully treated in this way it is impossible to ascertain owing to the absence of confirmation of the diagnosis.

All the severer cases fail to respond to conservative treatment, and the opinion prevalent among most observers appears to be that after the onset of acute symptoms the sooner operative measures are undertaken the better is the prognosis. It is true that operative treatment hitherto has not been encouraging in its results, but it is to be remembered that in many cases the patient was practically moribund before the surgeon could intervene, and that in some the infarct involved a larger segment of the intestine than can be removed under any circumstances.

An analysis is given below of different types of operation that have been performed.

In the following cases the *abdomen was simply explored*, and nothing further could be done:—

Bewley¹, Bradford, Cartby², Cramer, Grosskurth³, C. A. Porter (2 cases)⁴, Greenough⁴, van Hook¹, de Jong, Kiliani, Köster⁵, Lagane, London Hosp. (3 cases), Lothrop², Monnier and Suter⁶,

¹ Revd. by Jackson, Porter and Quinby.

² „ „ Neutra.

³ „ „ Talke.

⁵ „ „ Kölbing.

⁷ „ „ Roussel.

⁹ „ „ Zesas.

⁴ Published by Jackson, Porter and Quinby.

⁶ Revd. by Maclaure and Jacoulet.

⁸ Revd. by Kiliani.

¹⁰ „ „ Sauvé.

Munro, Niederstein, original case No. 1, Péron and Beaussenat⁷, Picqué and Grégoire³, Picot, Sachs⁵, Schnitzler, G. M. Smith, Sprengel, Talke, and Woolsey⁸.

Total, 30 cases, all of which proved fatal.

An *artificial anus only* was made in 10 cases:—

Borszéky, Deckart (2 cases), Elliot, Harrington⁴, Mignon and Dopter, Picqué and Grégoire³, Polya, Senni⁶, and Whipple. In Senni's case the patient recovered.

Drainage of the abdomen was carried out in the following cases:—

Fischer⁶, London Hosp. (1 case), Mumford⁴, original case No. 5, Thorndike⁴, and Tschudy⁶. Two of these cases recovered, viz. that reported by Tschudy, and original No. 5. In the former the infarcted caecum was packed off from the rest of the peritoneal cavity, and in the latter a coil of ileum shewing extreme engorgement became attached repeatedly to the pelvic wall and had to be freed.

The *veins in the ileo-caecal angle were drained* in two cases by Gerster. Both patients recovered.

The *infarcted loop of intestine was 'exteriorised'* in four cases, none of which survived. They were reported by Bloodgood⁴, M'Carthy, Maclaure and Jacoulet, and Mouchet⁹. The affected intestine in each case was fixed outside the abdomen for purposes of observation.

Resection of infarcted intestine has been carried out in a large number of cases which may be tabulated according to the manner of treating the divided ends. (Recoveries are indicated by the letter 'R' after the observer's name.)

End-to-end anastomosis was performed in the following:—

Codman (R), Gordon (R), Gosset¹⁰ (R), Kendal-Franks, Lecène¹⁰, Lewis and Rosenow, original case No. 3, C. B. Porter⁴, Shoemaker (R), Tcherkavskaja.

Total, 10 cases including 4 recoveries. In the last case *ileo-sigmoidostomy* was effected as well as end-to-end anastomosis.

End-to-end anastomosis with Murphy's 'button' was effected in the following cases:—

Brunner (R), Greensfelder, Neumann¹⁰ (R), Patal¹⁰ (R), Polya,

Schley (R), Sprengel, Stéphan⁶, Talke, Tyson and Livingston, Wakefield (R).

Total, 11 cases including 5 recoveries.

Lateral anastomosis was carried out in the following:—

Childe (R), Haagen⁶ (R) who used Murphy's 'button,' Lagane, Lindner⁹, original case No. 7, Straton.

Total, 6 cases including 2 recoveries.

The *divided ends were brought out of the abdomen* in the following:—

Codman, Elliot (R) who did an entero-anastomosis later, Greenough⁴ (upper end only brought out), Kanis, Körte¹⁰, Lerat and Cleret, Mitchell⁴, C. A. Porter⁴, Sprengel (R).

Total, 9 cases including 2 recoveries.

Gastro-jejunosomy was performed after resection, and followed by recovery in Kölbing's case.

Ileo-colostomy by means of Murphy's 'button' was carried out after resection in Leclerc and Cotte's case, but the patient died.

Ileo-colostomy by suture was performed after resection in three cases:—Sauvé (Lenormant's case), Gobiet (R), and von Baracz who also '*exteriorised*' the anastomosis.

Anastomosis was presumably carried out in six other cases:—

Blake, Jenni (R) (reported by Taravellier), de Jong, Kendal-Franks, Monnier and Suter⁶, Thévenot and Rey.

To summarise the operative results, if we exclude those 30 cases in which nothing radical was attempted we find that there were 25 cures out of 69 interventions, giving a percentage of 36·2 cures and 63·8 deaths.

The 69 cases in which radical treatment was attempted may be tabulated in the following way:—

Arterial oclusions	...	...	...	...	19	cases with	7	cures
Venous oclusions	...	...	...	...	40	"	"	13 "
Either arterial or venous oclusions	...			...	3	"	"	1 cure
Simultaneous arterial and venous oclusions					7	"	"	4 cures
					69	"		25 "

When operative interference is under consideration the important question of the kind of *anaesthetic* to be administered has to be settled. Gobiet is strongly opposed to the use of a general anaesthetic of any description on account of the consequent depression in blood-pressure already dangerously lowered by the internal haemorrhage. In its place he recommends the adoption of medullary anaesthesia, though admitting that it is by no means free from danger (e.g. respiratory failure, paraplegia). He used this method in his own case, the drug employed being tropacocaine (·06 grm.): the patient was pulseless throughout the operation which took $1\frac{1}{4}$ hours, but improved later under the influence of saline infusions and cardiac stimulants. Gobiet's view is probably correct as regards all the older methods of administering general anaesthetics. Chloroform in any form should be avoided; but ether by the 'open' method, preceded by atropine hypodermically, may be used with less danger in some cases, particularly if an intravenous saline infusion is given during the operation.

Spinal analgesia is a method very much in favour for acute abdominal cases, partly on account of the comparative absence of shock to the patient, and the absence of severe after effects, such as post-anaesthetic vomiting, acidosis, etc. and partly on account of the complete relaxation of the abdominal muscles thereby obtained.

But there is very little doubt that the best anaesthetic for these cases where the patient is much collapsed is the infusion into a vein of a 5 per cent. solution of ether in normal saline, a form of anaesthetic first adopted in Germany by Burkhardt and Kümmel, and introduced into this country by Rood in 1911. This method combines the use of a relatively safe drug with the administration of a continuous saline infusion, and the relaxation of the abdominal muscles is equalled only by the method of spinal analgesia. The infusion method may be preceded by a hypodermic of atropine or atropine and morphine.

The details of technique in intestinal resections will not be considered here as they belong rather to the province of the surgeon: but it will not be unprofitable to consider some of the dangers to be avoided.

An important matter to be borne in mind in the resection of

a portion of the small intestine is not so much the length of bowel removed as the amount left behind. Supposing less than a certain amount is left and the patient recovers from the shock of the operation, symptoms of inanition often associated with intractable diarrhoea supervene, and death follows from exhaustion. Zesas quotes experiments on animals in which one half the small intestine was removed without danger, and concludes that it is not prudent in man to leave less than two-thirds (i.e. 5 metres) behind¹. Von Baracz resected 350 cms. (including a small portion of colon) and the patient died of inanition. Gobiet on the other hand resected 320 cms. and the patient survived: it is to be noted however that his patient suffered from severe diarrhoea for months afterwards².

Another matter to be considered is the nature of the causal lesion. If infarction is due to venous thrombosis the probability is that the actual infarcted area represents only a small part of the region containing thrombosed veins, and that after resection the process will spread. For this reason the plan has been adopted by some operators of bringing the intestine above and

¹ See also Flint's work in section above ('Experiments on animals').

² Flint collected a series of 59 cases in which extensive resections of the small intestine had been carried out for various conditions. In all but one case 200 cms. and over were removed, and out of the whole series 50 patients recovered. To quote a few of the cases:—Ruggi removed 330 cms. of small intestine from a boy of 8 'in three sittings.' After the third operation the nitrogen and fat in the faeces were at first normal, but increased later. Fantino resected 310 cms. of combined jejunum and ileum in a patient of 60. In spite of a high percentage of nitrogen and fat in the faeces after resection the patient continued to gain in weight, and made an uninterrupted recovery. Nigrisoli resected 520 cms. of small intestine. This was followed by an increase of nitrogen and fat in the faeces, and of intestinal putrefaction (indicated by an increase of ethereal sulphates in the urine). A patient of Brenner's, after the removal of 540 cms. of ileum, died 2½ years after from marasmus. The health had been good apparently in the meantime though at first the patient had had five liquid offensive stools daily. The patient was a woman of 61.

Flint says that the resection of large amounts of the small intestine, by diminishing the absorbing surface of the gut, disturbs the balance in the nitrogen and fat absorptions, and leads to an excessive 'excretion' of these elements in the faeces, an increase which may reach two-thirds of the quantity ingested. This accounts for the profuse diarrhoea invariably observed after the operation. The loss of weight is accounted for by the consumption of tissue nitrogen and fats. Gradually the balance is restored, and with the exception of excessive intestinal putrefaction, the normal conditions may be recovered.

below the anastomosis outside the abdominal wall at the end of the operation for the purpose of subsequent observation.

Though a spreading of the infarcted zone is not so likely to take place in an arterial lesion, another difficulty arises, viz. the setting free of further emboli from a diseased heart or aorta. There is unfortunately no way of preventing this, though the risks may be minimised by keeping the patient as quiet as possible after the operation. This is one reason why special care must be taken in the choice of the anaesthetic.

In certain cases of appendix abscess where it is impossible or not prudent to remove the appendix when the abscess is drained, Wilms¹ has suggested an operative measure to minimise the risk of mesenteric thrombo-phlebitis and hepatic abscess. The case in which it was first tried may be briefly summarised:—

M. 42. Large abscess that took ten days to develop in the appendix region. Abscess opened by Wilms. On 2nd day after, pt. had a rigor; on 3rd day three severe rigors; T. 41·2, 40·8, and 40·3; on 4th day, rigor; T. 40·4; two hours after the rigor, operation. Horizontal skin incision rather above level of ant. sup. iliac spine extending to margin of rectus. Finger placed beneath lower end of asc. colon, and this with its mesentery and that of lower end of ileum separated from post. abdominal wall; anterior peritoneal layer of mesentery separated; arteries in mesentery held to one side, and veins ligatured in two bundles; tampon placed on caecum in case of circulatory disturbances and gangrene; drainage. The result was that there was no further rise of temperature, and rigors ceased. Wilms believes that by cutting off the venous return from the highly septic focus (caecum, appendix, and lower ileum) to the mesenteric veins thrombo-phlebitis was prevented.

Sprenkel² tried this form of treatment but was not so fortunate. Anatomical difficulties prevented satisfactory access to the veins, which were tied nevertheless, and circulatory changes led to local necrosis in the wall of the caecum. The patient died with thrombo-phlebitis of both mesenteric veins, and multiple abscesses in the liver. Commenting upon Wilms' operation Sprenkel

¹ Wilms, *Zentralblatt für Chirurgie*, 1909, Nr. 30, S. 1041.

² Sprenkel, *Zentralblatt für Chirurgie*, 1911, Nr. 2, S. 33.

emphasises the grave dangers that exist of infecting the retro-peritoneal tissues and of producing gangrene of the caecum and appendix, together with the difficulties of freeing the caecum if bound down by adhesions or by peritoneum, and of obtaining access to the vessels in a fat patient.

The theoretical objections to Wilms' operation seem to be insurmountable; for either the venous return from the ileo-caecal region is completely shut off, with the result that infarction follows, or the septic matter in the veins is diverted into the caval system instead of into the portal. It is scarcely probable that a continuous venous hyperaemia can have any curative action upon a highly septic focus, containing may be a gangrenous appendix. Finally, the dangers that attend the modern operation of removing the appendix are probably a great deal less than those associated with Wilms' operation.

Gerster's operation of curetting and draining the infected veins at the ileo-caecal angle is safer and less difficult to carry out than Wilms' procedure, but it seems clear that whatever method is adopted, there will always remain the danger of thrombosis or pyelephlebitis so long as the appendix is present. Once the diseased appendix has been removed the main source of infection ceases to exist, and the possibility of the supervention of thrombo-phlebitis in cases of appendicitis is only another argument in favour of early removal in all cases¹.

PROGNOSIS.

Although we have to a certain extent dealt with prognosis in the discussion of treatment, yet a few words must be said regarding the cases that are never treated surgically. In considering these cases we are confronted with the same difficulty as when discussing the efficacy of conservative methods of treatment. That is to say, apart from operation or post mortem examination it is impossible to be certain in the present state of our knowledge that any given

¹ In this connection Polya's opinion may be quoted:—

‘Diese Komplikation der Appendicitis ist, da die Eiterherde in dem Stamm der Vena mesaraica unangreifbar sind, als eine fast sicher tödliche zu betrachten, und die chirurgische Therapie liegt hauptsächlich in der Prophylaxe—nämlich, der frühzeitigen Entfernung des erkrankten Wurmfortsatzes.’ (*Loc. cit.* S. 344.)

case is one of mesenteric vascular occlusion. The clinical circumstantial evidence may be strong, but it is never certain.

We have already had occasion to refer to cases¹ where an efficient collateral circulation had been established. Other cases that did not prove fatal, but in which the evidence of mesenteric vascular occlusion was very strong, were reported by Finlayson, Cohn, Karcher, Moos, and Ott.

Two cases² are on record in which there was definite evidence of old vascular obstruction in portions of the mesentery; in one of these there was stenosis of the intestine, while in the other there were puckering of the mesentery and wide anastomosing channels.

All these cases however must be regarded as most exceptional. The disease nearly always proves fatal when any but the smallest mesenteric vessels are occluded, unless operative measures are undertaken. The prognosis after operation is practically the same whether the causal occlusion affected the arteries, the veins, or both the arteries and the veins. Thus cures were effected in 36·8 % of cases of arterial occlusion, in 32·5 % of venous, and in 36·2 % of arterial and venous occlusions together.

¹ Virchow, Chiene, Original case No. 6.

² Langerhans (revd. by Talke), Packard (revd. by Mauclaire and Jacoulet).

SECTION IX

CONCLUSIONS

(1) MESENTERIC vascular occlusion is not an extremely rare disease; about 360 cases of this nature have been collected as a foundation for the present work¹.

The disease very rarely affects children: adults between the ages of 30 and 70 are most liable to be attacked. Males are affected in about 62 %, and females in 38 % of cases.

(2) The effect of mesenteric vascular occlusion was haemorrhagic infarction of the intestine in about 64 % of the undoubted cases in the present series. When infarction did not occur, extreme engorgement, ulceration, perforation or changes of minor severity were observed with few exceptions in the area of supply or drainage of the occluded vessels.

Anaemic infarction occurred in 2·7 % of the cases.

(3) The vascular occlusion may affect the arteries, the veins, or both arteries and veins together. The arteries or their branches were occluded in 53 % of the cases, the veins or their tributaries in about 41 %, and arteries and veins together in about 6 %.

In the vast proportion of cases the superior mesenteric vessels were involved, and not the inferior.

(4) In the present series the most common cause of arterial occlusion was embolism. This originated in most cases from acute or chronic endocarditis following or associated with rheumatic fever, and less frequently from thrombi in the heart, and atheroma of the aorta.

¹ *Vide infra* 'List of cases.'

In a few cases malignant endocarditis was responsible for the embolic process.

The commonest cause of thrombosis of the arteries was embolism. Much less frequently thrombosis was caused by atheroma of the aorta, of the mesenteric artery, or arterio-sclerosis and endarteritis of the branches of the artery.

Venous thrombosis in the mesentery most commonly arises from the downward spread of thrombosis from the portal vein. In other cases it arises primarily in the mesenteric system as a result of a septic process in the appendix, or in the female pelvic organs.

(5) In the great majority of cases the disease runs a course lasting from a day or two up to ten or fourteen days. When the intestinal lesion is due to an arterial obstruction it tends to be more acute than when it is due to venous obstruction. 82 % of the cases resulting from combined arterial and venous occlusions in this series survived for a period not exceeding seven days.

When large or medium sized vessels are occluded the disease nearly always ends fatally, unless the patient is operated upon at an early stage.

(6) The most striking manifestations of the disease are the sudden onset, the acute abdominal pain, vomiting and diarrhoea, the stools and vomit not uncommonly containing blood. Constipation is often a prominent feature. Shock is generally severe: the pulse becomes small, very rapid and irregular, the temperature is normal or subnormal but often becomes raised at an early stage. Distension of the abdomen is progressive, though it is not extreme in the initial stage of the disease. An abdominal tumour has been observed in about 5.6 % of cases of infarction.

Visible peristalsis is extremely rare in cases of mesenteric vascular occlusion.

Not uncommonly after the onset the pain, which is sometimes agonising, shews some temporary diminution of intensity, though the other symptoms continue unabated till death.

(7) Peritonitis complicated the disease in at least 28 % of the cases in this series.

(8) Treatment. Operation should be undertaken as soon as possible after the onset of the acute symptoms. If infarction has occurred resection is the only measure that is likely to meet with success. In cases of venous thrombosis resulting from sepsis in the appendix region or female pelvic organs the septic focus should if possible be removed.

If the affected intestine is still viable, and the vascular lesion is not extending, recovery may take place without resection.

The results of operation, exclusive of cases in which nothing was attempted, give 36·2 % of recoveries.

It is probable that in some early cases the removal of the primary septic focus may check the extension of the thrombosis.

BIBLIOGRAPHY

- ABRAM. 'Coincident embolism of cerebral, mesenteric and femoral arteries.' *British Medical Journal*, 1907, Vol. 2, p. 1465.
- AKIN. 'Gastric symptoms consequent on arterio-sclerosis.' *Journal of American Medical Association*, 1909, Vol. 52, p. 1825.
- ALCANUS. 'Ein Fall von Embolie der Arteria mesaraica superior.' *Münchener Medicinische Wochenschrift*, 1903, S. 491.
- ALBUTT AND ROLLESTON. *System of Medicine*, Vol. 3, London, 1907.
- ALLEN. 'Embolism of the superior mesenteric artery after normal labour.' *Lancet*, 1900, Vol. 1, p. 1074.
- ALTMANN. 'Ein Fall ausgedehnter hämorrhagischer Infarcierung des Darmes durch thromboembolische Prozesse in der Arteria mesaraica superior.' *Virchows Archiv*, 1889, Bd. 117, S. 206.
- AMOS. 'Zur Pathologie der Thrombose der Vena mesaraica superior.' *Zeitschrift für Geburtshilfe und Gynäkologie*, 1905, Bd. 55, S. 430.
- ASKANAZY. *Revue Médicale de la Suisse Romande*, 1908, pp. 156 et 437.
- AUVRAY. 'Un case d'infarctus hémorragique de l'intestin par thrombose veineuse mésentérique.' *Bull. et mém. de la Soc. de Chir. de Paris*, T. 35, p. 138. (Revd. in *Centralblatt für Chirurgie*, 1910, S. 59.)
- BALLANCE. 'A case of hæmorrhagic infarction of the small intestine due to thrombosis of the superior mesenteric vein.' *Lancet*, 1899, Vol. 2, p. 85.
- v. BARACZ. 'Brucheinklemmung complicirt durch Thrombose der Vena mesaraica superior.' *Archiv für Klinische Chirurgie*, 1907, Bd. 83, S. 478.
- BARDY. 'Beitrag zur Kenntniss des Darminfarktes.' *Finska Lakarisällsk.*, Hand 1, Bd. 52, Nr. 3. (Revd. in *Centralblatt für Chirurgie*, 1910, S. 1178.)
- BECKMANN. 'Embolie der Arteria mesaraica superior.' *Virchows Archiv*, 1858, Bd. 13, S. 501.
- BIER. 'Die Entstehung des Collateralkreislaufs.' (Theil 1, 'Der arterielle Collateralkreislauf. 2.')
- Virchows Archiv*, 1897, Bd. 147, Se. 256 und 444.
- BLAKE. *Annals of Surgery*, 1906, Vol. 1, p. 614.
- BOLOGNESI. 'Der Verschluss der Mesenterialgefässe.' *Virchows Archiv*, 1911, Bd. 203, Heft 2, S. 213.
- BOOTH. 'Ulcerative endocarditis; anomalous skin eruption; intestinal ulceration; death, necropsy.' *Lancet*, 1886, Vol. 1, p. 785.

- BORRMANN. 'Beiträge zur Thrombose des Pfortaderstammes.' Deutsches Archiv für Klinische Medizin, 1897, Bd. 59, Heft 3 und 4, S. 283.
- BORSZÉKY. 'Ileus durch Embolie der Arteria mesenterica superior.' Beiträge zur Klinischen Chirurgie (von Bruns), 1901, Bd. 31, Heft 3, S. 704.
- BRADFORD. 'A case of thrombosis of the superior mesenteric vein, causing intestinal obstruction.' Transactions of Clinical Society, London, Vol. 31 (Oct. 1897 to May 1898), p. 203.
- BREWER. Annals of Surgery, 1908, Vol. 1, p. 619.
- BRUNNER. 'Ein durch Operation geheilter Fall von septischer Thrombose der Mesenterialvenen.' Deutsche Zeitschrift für Chirurgie, 1907, Bd. 89, S. 624.
- BURGESS. 'A case of thrombosis of the superior mesenteric vein.' Dublin Journal of Medical Science, Feb. 1911, pp. 114 and 145.
- BURROWS. 'Hydatid of the liver, and ligature of the portal vein. Recovery.' Annals of Surgery, 1908, Vol. 1, p. 619.
- BUTLIN. 'Growth in the left ventricle, with embolism in the brachial and other arteries.' British Medical Journal, 1879, Vol. 2, p. 657.
- CHIENE. 'Complete obliteration of the coeliac and mesenteric arteries: the viscera receiving their blood-supply through the extraperitoneal system of vessels.' Journal of Anatomy and Physiology, 1869, p. 65.
- CHILDE. 'A case of successful resection of $9\frac{1}{2}$ feet of small intestine for gangrene.' Lancet, 1907, Vol. 2, p. 379.
- CLAISSE ET ABRAMI. Société Médicale des Hôpitaux, 1905, p. 349.
- CODMAN. 'A case of mesenteric thrombosis; resection of intestine; end-to-end anastomosis. Recovery.' Boston Medical and Surgical Journal, March 17th, 1910, p. 355.
- COLLINS. 'Circulatory disturbances of the mesenteric vessels.' New York Medical Journal, June 10th, 1911, p. 1127.
- COUNCILMAN. 'Three cases of occlusion of the superior mesenteric artery.' Boston Medical and Surgical Journal, 1894, Vol. 1, p. 410.
- CRAMER. 'Embolie der Arteria mesenterica superior im Puerperium.' Münchener Medicinische Wochenschrift, 1904, Nr. 46, S. 2049.
- CURTIS AND LAWRENCE. 'A case of portal thrombosis.' Lancet, 1909, Vol. 1, p. 910.
- DATE. 'A case of mesenteric embolism.' British Medical Journal, 1904, Vol. 1, p. 725.
- DECKART. 'Ueber Thrombose und Embolie der Mesenterialgefäße. Ein Beitrag zur Lehre vom Ileus.' Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1899, Bd. 5, Heft 3, S. 511.
- DELATOUR. 'Thrombosis of the mesenteric veins: a cause of death after splenectomy.' Annals of Surgery, 1895, Vol. 1, p. 24.
- DOUGLAS. Surgical diseases of the abdomen. London, 1909.
- DOWDEN. 'Case of acute gangrene of the caecum, ascending, transverse and descending colon. Operation. Death. Autopsy.' Edinburgh Medical Journal, May 1911, p. 435.

- DUNLOP. 'A case of thrombosis of the superior mesenteric veins.' *Lancet*, 1900, Vol. 1, p. 1800.
- ELLIOT. 'Operative relief of gangrene of the intestine due to occlusion of the mesenteric vessels.' *Annals of Surgery*, 1895, Vol. 1, p. 9.
- FABER. 'Die Embolie der Arteria mesenterica superior.' *Deutsches Archiv für Klinische Medizin*, 1875, Bd. 16, S. 527.
- FAGGE. 'A case of thrombosis of the superior mesenteric and portal veins attended by rapidly fatal collapse.' *Transactions of Pathological Society*, London, 1875—1876, p. 124.
- FEDOROWITSCH. 'Zur Frage vom Verschluss der Mesenterialgefäße.' *Arbeiten der propädeut.-chirurg. Klinik von Prof. W. A. Oppel*, Bd. 11, 1910. (Russisch.) (Revd. in *Centralblatt für Chirurgie*, 1911, Nr. 17, S. 625.)
- FINLAYSON. 'Report of a case of embolism of the superior mesenteric artery.' *Glasgow Medical Journal*, 1888, p. 414.
- FLINT. 'The effect of extensive resections of the small intestine.' *Bulletin of Johns Hopkins Hospital*, May 1912, p. 127.
- GALLAVARDIN. 'Embolies et thromboses des vaisseaux mésentériques (oblitérations des artères mésentériques et thrombo-phlébites mésentériques).' *Gazette des Hôpitaux*, 1901, pp. 929 et 957.
- GARMSEN. 'Ein Fall von haemorrhagischer Infarcirung des Dünndarms nach Thrombose der Pfortader.' *Inaugural-Dissertation*, Kiel, 1895.
- GERSTER. 'On septic thrombosis of the roots of the portal vein in appendicitis, and on pyelephlebitis, together with some remarks on peritoneal sepsis.' *Medical Record*, 1903, Vol. 1, p. 1005.
- GILLETT. 'Infaret of the transverse colon.' *Lancet*, 1911, Vol. 2, p. 220.
- GOBIET. 'Ueber Verschluss der Mesenterialgefäße nebst Mitteilung eines operativ geheilten Falles.' *Wiener Klinische Wochenschrift*, 1911, Nr. 45, S. 1552.
- GOODHEART. 'Arterio-venous aneurism of the splenic vessels with thrombosis of the mesenteric veins and localised acute colitis.' *Transactions of Pathological Society*, London, 1889, Vol. 40, p. 67.
- GORDON. 'A case of haemorrhagic infarction of the small intestine: successful resection.' *British Medical Journal*, 1898, Vol. 1, p. 1447.
- GOULD. 'Certain diseases of the blood-vessels.' *Lancet*, 1902, Vol. 1, p. 721.
- GRAWITZ. 'Fall von Thrombose der Pfortader.' *Virchows Archiv*, 1887, Bd. 110, S. 439.
- GREENSFELDER. 'Thrombosis of the superior mesenteric artery.' *Annals of Surgery*, 1905, Vol. 1, p. 302.
- HANDFORD. 'Thrombosis of portal and mesenteric veins and of the vena cava.' *Lancet*, 1892, Vol. 2, p. 91.
- HEGAR. 'Enucleation eines grossen intraparietalen Myoms. Marantische Thrombose der linken Schenkelvene. Embolie der linken Arterie und der Arteria mesaraica inferior.' *Virchows Archiv*, 1869, Bd. 48, S. 332.

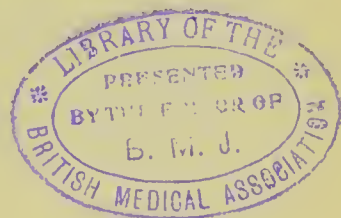
- HIRTZ ET JOSUÉ. 'Thrombophlébite de la veine porte et des veines mésentériques.' Bulletin de la Société Médicale des Hôpitaux, 1900, T. 17, p. 872.
- HUBSCHMANN. Revue Médicale de la Suisse Romande, 1908, p. 544.
- JACKSON, PORTER AND QUINBY. 'Mesenteric embolism and thrombosis. A study of two hundred and fourteen cases.' Journal of American Medical Association, 1904, June 4, July 2, 9 and 16.
- DE JONG. 'Ueber die Folgen der Thrombose im Gebiete des Pfortadersystems.' Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1911, Bd. 24, Heft 1, S. 160.
- JUDD. Journal of American Medical Association, 1907, Vol. 49, p. 1842.
- KADER. 'Ein experimenteller Beitrag zur Frage des localen Meteorismus bei Darmocclusion.' Deutsche Zeitschrift für Chirurgie, 1892, Bd. 33, Heft 1, 2 and 3, Se. 57 und 214.
- KANIS. Wiener Klinische Rundschau, 1911, Nr. 48 und 49. (Revd. in British Medical Journal, 1912, Vol. 1, epitome No. 162, p. 45.)
- KARCHER. 'Ein Fall von Embolie der Arteria mesenterica superior.' Correspondenzblätter für Schweizer Aerzte, 1897, Nr. 18, S. 548.
- KAUFMANN. 'Ueber den Verschluss der Arteria mesaraica superior durch Embolie.' Virchows Archiv, 1889, Bd. 116, S. 353.
- KEMP. Diseases of the Stomach and Intestines. London, 1910.
- KENDAL-FRANKS. Transactions of Royal Academy of Medicine in Ireland, 1893, Vol. 11, p. 249.
- KILIANI. 'Thrombosis of the superior and inferior mesenteric artery.' Annals of Surgery, 1903, Vol. 2, p. 110.
- KÖLBING. 'Die Thrombose der Mesenterialgefäße.' Beiträge zur Klinischen Chirurgie (von Bruns), 1902, Bd. 33, Heft 2, S. 518.
- LAGANE. 'Les artérites intestinales : étude clinique, anatomo-pathologique et expérimentale.' Thèse, Paris, Dec. 1911.
- LAGANE. 'Le Syndrome artério-scléreux de l'intestin.' Presse Médicale, Dec. 13, 1911, p. 1025.
- LECLERC ET COTTE. 'Oblitération des vaisseaux mésentériques avec infarctus et gangrène anémiques de l'intestin.' Lyon Chirurgial, Mai 1911, T. 5, No. 5, p. 483.
- LEECH. Quarterly Medical Journal for Yorkshire and Adjoining Counties, 1898, Vol. 6, p. 370.
- LEWIS AND ROSENOW. 'Primary portal thrombosis.' Surgery, Gynaecology and Obstetrics, Chicago, 1898, Vol. 2, p. 586.
- LISSAUER. 'Beitrag zur Frage der Entstehung der Pfortaderthrombose.' Virchows Archiv, 1908, Bd. 192, Heft 2, S. 278.
- LITEN. 'Ueber die Folgen des Verschlusses der Arteria mesenterica superior.' Virchows Archiv, 1875, Bd. 63, S. 289.
- LOCKWOOD. 'Wound of the mesentery with subsequent gangrene of the intestine.' Lancet, 1897, Vol. 1, p. 1413.
- LONDON HOSPITAL POST-MORTEM RECORDS. 1907, Nos. 565, 640, 699, 827,

- 1120, 1133 ; 1908, Nos. 45, 330, 603, 879, 1144, 1153, 1230 ; 1909, Nos. 529, 813, 986 ; 1910, Nos. 123, 136, 458, 500, 715, 802, 903, 931, 997 ; 1911, Nos. 50, 119, 147, 154, 316.
- MCARTHUR. 'Ileus due to vascular obstruction.' *Annals of Surgery*, 1901, Vol. 1, p. 443.
- MCCARTHY. 'Acute intestinal obstruction probably due to embolism of several branches of the superior mesenteric artery.' *Lancet*, 1890, Vol. 1, p. 646.
- MACPHERSON. 'A case of thrombosis of the mesenteric veins.' *British Medical Journal*, 1905, Vol. 2, p. 330.
- M'WEENEY. 'Thrombosis of the superior mesenteric vein.' *Lancet*, 1893, Vol. 2, p. 1576.
- MANASSE. 'Die arterielle Gefässversorgung des S. romanum in ihrer Bedeutung für die operative Verlagerung desselben.' *Archiv für Klinische Chirurgie*, 1907, Bd. 83, S. 999.
- MAREK. 'Ueber die Folgen des Verschlusses der Gekrösarterien.' *Deutsche Zeitschrift für Chirurgie*, 1907, Bd. 90, Heft 1, 2 und 3, S. 174.
- MAUCLAIRE ET JACOULET. 'L'infarctus hémorrhagique de l'intestin par oblitération veineuse ou artérielle.' *Archives Générales de Chirurgie*, 1908.
- MAYGRIER ET LETULLE. 'Thrombophlébite puerpérale de la veine mésentérique : apoplexie veineuse partielle de l'intestin grêle.' *Bulletins de la Société Anatomique*, 1898, p. 507.
- MAYLARD. 'Two cases of post-operative thrombosis of the mesenteric vessels followed by death.' *British Medical Journal*, 1901, Vol. 2, p. 1454.
- MERKEL. 'Ueber Verschluss der Mesenterialarterien und dessen Folgen.' *Münchener Medizinische Wochenschrift*, Dec. 5, 1911, S. 2604.
- MEYER. 'Un cas d'infarctus anémique de l'intestin grêle.' *Charkovsky Meditsinsky Journal*, Tome 12, No. 5, Dec. 1911, p. 508. (Revd. in *Journal de Chirurgie*, Fév. 1912, Tome 8, No. 2, p. 180.)
- MICHAELIS. 'Zur Casuistik der Embolie der Darmarterien.' *Virchows Archiv*, 1899, Bd. 156, S. 181.
- MIGNON ET DOPTER. 'Thrombo-phlébite mésentérique primitive.' *Presse Médicale*, Oct. 29, 1904, No. 87, p. 689.
- MONRO AND WORKMAN. 'Thrombosis of the mesenteric veins in enteric fever.' *Glasgow Medical Journal*, 1900, Vol. 54, p. 294.
- MOOS. 'Beitrag zur Casuistik der embolischen Gefässkrankheiten.' *Virchows Archiv*, 1867, Bd. 41, S. 58.
- MOYNIHAN. *Abdominal Operations*. London, 1906.
- MUNRO. 'Embolism of the mesenteric vessels with symptoms of obstruction. Abdominal section.' *Lancet*, 1894, Vol. 1, p. 147.
- NEUTRA. 'Ueber die Erkrankungen der Mesenterialgefässe und ihre Bedeutung für die Chirurgie.' *Centralblatt für die Grenzgebiete der Medizin und Chirurgie*, 1902, Bd. 5, Nr. 18, 19, 20, 21 und 22, S. 705, 737, 785, 830 und 865.

- NIEDERSTEIN. 'Zirkulationsstörungen im Mesenterialgebiet.' Deutsche Zeitschrift für Chirurgie, 1906, Bd. 85, S. 710.
- NIEDERSTEIN. 'Zirkulationsstörungen im Mesenterialgebiet.' Deutsche Zeitschrift für Chirurgie, 1909, Bd. 98, S. 188.
- NUTT. 'A case of intestinal infarction.' British Medical Journal, 1903, Vol. 2, p. 1273.
- ORTHNER. 'Zur Klinik der Angiosklerose der Darmarterien (Dyspragia intermittens angiosclerotica intestinalis) nebst einem Beiträge zur Klinik des intermittirenden Hinkens und des Stokes-Adam'schen Symptomenkomplexes.' Sammlung Klinischer Vorträge (von Volkmann), Jan. 1903, Nr. 347.
- OSLER. Medical News, 1883, Vol. 42, p. 693.
- OSSWALD. 'Zur Casuistik der Embolie und Thrombose der Mesenterialgefäße.' Zeitschrift für Klinische Medizin, 1904, Tome 53, S. 308.
- PAYR. 'Experimente über Magenveränderungen als Folge von Thrombose und Embolie im Pfortadergebiete.' Archiv für Klinische Chirurgie, 1907, Bd. 84, S. 799.
- PICOT. 'Un cas de thrombose des veines mésentériques pris pour une colique hépatique.' Revue Médicale de la Suisse Romande, 20 Août, 1908, p. 537.
- POLYA. 'Thrombophlebitis mesaraica, eine verhängnisvolle Komplikation der Appendicitis.' Deutsche Medizinische Wochenschrift, Feb. 16, 23, und März 2, 1905, Se. 257, 299 und 341.
- REINECKE. 'Isolierte, quere Mesenterialabreissung bei Kontusion des Abdomens.' Wiener Klinische Wochenschrift, 1908, S. 1605.
- REITZENSTEIN. 'Ileus infolge von Thrombose der Vena mesenterica.' Münchener Medizinische Wochenschrift, 1903, S. 257.
- RICHARDS. 'Gangrene of the intestine from partial obstruction of the superior mesenteric artery.' British Medical Journal, 1905, Vol. 2, p. 1521.
- ROBSON. 'A case of perforating wound of the abdomen in which the superior mesenteric vein was wounded.' British Medical Journal, 1897, Vol. 2, p. 77.
- RODGER. 'Coincident embolism of cerebral, mesenteric and femoral arteries.' British Medical Journal, 1907, Vol. 1, p. 810.
- ROLLESTON. 'Thrombosis of the splenic vein associated with infarcts.' Pathological Society, London, Jan. 19th, 1892; Lancet, 1892, Vol. 1, p. 196.
- ROLLESTON. Diseases of the liver, gall-bladder and bile-ducts. London, 1905.
- ROLLESTON. System of Medicine, Allbutt and Rolleston, Vol. 3. London, 1907.
- ROOD. 'Ether infusion anaesthesia.' Proceedings of Royal Society of Medicine, London, 1912, Vol. 5, No. 6, p. 77.
- ROPER. 'A case of mitral stenosis: death from embolism of six main arteries.' Lancet, 1906, Vol. 2, p. 439.

- ROUGHTON. 'A case of ruptured mesenteric artery : laparotomy : recovery.' *Lancet*, 1899, Vol. 1, p. 89.
- ROUSSEL. 'Contribution à l'étude de l'infarctus hémorrhagique de l'intestin consécutif aux thrombo-phlébites mésentériques.' Thèse, Paris, 1905.
- SAUVÉ. 'De l'oblitération des vaisseaux mésentériques (infarctus de l'intestin).' *Journal de Chirurgie*, Nov. 1910, Tome 5, p. 485.
- SCHLESINGER. *Münchener Medizinische Wochenschrift*, 1903, S. 140.
- SCHLEY. 'Mesenteric thrombosis. Operation. Recovery.' *St Luke's Hospital Medical and Surgical Reports*, New York, 1910. (Revd. in *Centralblatt für Chirurgie*, 1911, Juli 15, S. 980.)
- SCHNITZLER. 'Zur Symptomatologie des Darmarterienverschlusses.' *Wiener Medicinische Wochenschrift*, 1901, Nr. 11 und 12, Se. 506 und 567.
- SHOEMAKER. 'Resection of 5 feet of intestine for thrombosis of the mesentery.' *New York Medical Journal*, Dec. 4th, 1909, Vol. 90, No. 23, p. 1102. (Revd. in *Journal de Chirurgie*, 1910, Tome 4, p. 182.)
- SMITH, F. J. 'Inflamed vermiform appendix.' *Lancet*, 1894, Vol. 1, p. 151.
- SMITH, G. M. 'A case of mesenteric thrombosis.' *Bristol Medico-chirurgical Journal*, 1906, Vol. 24, p. 216.
- SMITH, W. G. 'Thrombosis of the portal system.' *Lancet*, 1894, Vol. 1, p. 155.
- SOLOWIEFF. 'Veränderungen in der Leber unter dem Einflusse künstlicher Verstopfung der Pfortader.' *Virchows Archiv*, 1875, Bd. 62, S. 195.
- SPIEGELBERG. 'Drei seltenere Sectionsbefunde : 1, Verkalkung der Wandungen der thrombotischen Pfortader.' *Virchows Archiv*, 1895, Bd. 142, S. 547.
- SPRENGEL. 'Zur Pathologie der Circulationsstörungen im Gebiet der Mesenterialgefässe.' *Verhandlungen der Deutsche Gesellschaft für Chirurgie*, 1902, Bd. 31, 'Grossere Vorträge,' S. 55.
- SPRENGEL. 'Zur Frage der "Venenunterbindungen bei eitriger Pfortaderthrombose nach Appendicitis"'. *Centralblatt für Chirurgie*, 1911, Nr. 2, S. 33.
- ST BARTHOLOMEW'S HOSPITAL MUSEUM CATALOGUE : SURGICAL. Nos. 1956 G, 1956 H, and 2205 A.
- STRATON. 'A case of thrombosis of the inferior mesenteric vein.' *Lancet*, 1910, Vol. 2, p. 311.
- TALKE. 'Ueber Embolie und Thrombose der Mesenterialgefässe.' *Beiträge zur Klinischen Chirurgie*, 1903, Bd. 38, Heft 3, S. 743.
- TANGL UND HARLEY. 'Notiz über einige anatomische Veränderungen nach Unterbindung der drei Darmarterien.' *Centralblatt für die Medicinische Wissenschaft*, 1895, Nr. 39, S. 673.
- TAYLOR. *Lancet*, 1881, Vol. 1, p. 56.
- TCHERKAVSKAÏA. 'Un case d'infarctus hémorrhagique de l'intestin grêle suite de thrombose d'une grosse branche de l'artère mésentérique supérieure.' *Chirurgia*, Tome 30, No. 180, Dec. 1911, p. 609. (Revd. in *Journal de Chirurgie*, 1912, Tome 8, No. 3, p. 311.)

- THÉVENOT ET REY. 'Thrombose de l'artère mésentérique supérieure simulant une tumeur abdominale.' *Archives Générales de Chirurgie*, 1911, Nov. 25, No. 11, p. 1234.
- TYSON AND LIVINGTON. 'A case of acute intestinal obstruction due to embolism of branches of the superior mesenteric artery.' *Lancet*, 1902, Vol. 1, p. 1248, and *British Medical Journal*, 1902, Vol. 1, p. 963.
- VIRCHOW. *Virchows Archiv*, 1847, Bd. 1, S. 332.
- VIRCHOW. *Verhandlungen der Physikalisch-medicinischen Gesellschaft in Würzburg*, 1854, Bd. 4, S. 341.
- WAKEFIELD, W. 'Thrombosis of the mesentery causing gangrene of the ileum.' *Journal of American Medical Association*, Oct. 1911, Vol. 57, No. 16, p. 1285.
- WEST. *Transactions of Pathological Society, London*, 1890, Vol. 41, p. 146, and *Lancet*, 1890, Vol. 1, p. 196.
- WHIPPLE. 'A case of intestinal obstruction due to thrombosis of some of the mesenteric vessels. Laparotomy, death, necropsy.' *Lancet*, 1900, Vol. 1, p. 705.
- WILMS. 'Zur Frage der Gefassverletzungen der Radix Mesenterii.' *Münchener Medicinische Wochenschrift*, 1901, S. 1277.
- WILMS. 'Venenerunterbindung bei eitriger Pfortaderthrombose nach Appendicitis.' *Centralblatt für Chirurgie*, 1909, Nr. 30, S. 1041.
- WOLOSCHIN. 'Zur Frage von der Embolie durch parenchymatöse (Leber-) Zellen.' *Wratschebnaja Gazeta*, 1909, Nr. 42. (Revd. in *Centralblatt für Chirurgie*, 1910, S. 67.)
- WOOD. 'Thrombosis and embolism of the superior mesenteric artery.' *Medical News*, 1883, Vol. 42, p. 637.
- ZESAS. 'Die Thrombose und Embolie der Mesenterialgefäße.' *Centralblatt für die Grenzgebiete der Medizin und Chirurgie*, Juni, 1910, Bd. 13.



LIST OF 366 CASES¹

- Adénot (N) 2, Alexander (N), Alkan (J, P & Q), Allen, Altmann, Amos, Anderson, Arnstein & Troschewsky (T), Aronshon (T), Askanazy 2, Aufrecht.
- Ballance, v. Baracz, Barth (J, P & Q), Bayer (T), Beckmann, Bewley (J, P & Q), Blake, Bonne (N), Booth, Bornhaupt (T), Borrmann, Borszéky, Bradford, Braquehayé (M & J), Breuer (J, P & Q), Broussin (M & J), Brunner, Bühner (T), Burgess, Burgess (J, P & Q), Butlin.
- Cartby (N), Cattani (N), Chiene, Childe, Chuquet (N), Claisse & Abrami, Codman, Cohn (N) 3, Concato (N), Conti (N), Councilman 3, Cramer, Curtis & Lawrence.
- Deckart 3, Delatour, Dinkler (T), Dreyfous (N) 2, Dreyfus (M & J), Drozda (T), Drozda (N) 2, Dun, Dunlop.
- Edler (T), v. Eicken (T), Eisenlohr (K), Elliot 2.
- Faber 4, Fagge, Falkenburg (J, P & Q) 2, Fedorowitsch 3, Finlayson, Firket & Malvoz (N), Fischer (M & J), Frankenhäuser (K), Fraser (J, P and Q), Fritz (J, P & Q).
- Gallavardin (T), Garmsen, Gerhardt (N), Gerster 2, Gimard (T), Ginsburg (J, P & Q) 3, Gintrac (N), Gobiet 2, Goodheart, Gordon, Gosset (S), Gould, Grawitz 2, Greensfelder, Grosskurth (T), Gull.
- Haagen (M & J), Hahn (N), Haliprè (J, P & Q), Handford, Hegar, Henderson, Hirtz & Josué, Högel (M & J) 2, Howse (N), Hübschmann.
- Jackson, Porter & Quinby 27² (J. M. Jackson, Gannett, J. J. Minot, R. H. Fitz 2, Bloodgood, Mitchell, Osler 2, C. A. Porter 3, C. B. Porter 2, M. H. Richardson 2, F. B. Harrington 2, R. B. Greenough 2, E. A.

¹ The publication of a single case is indicated by the name of the author; and a figure after an author's name signifies the number of original cases—if more than one was published.

In those instances where the original publications have not been accessible the initials of the reviewers of the cases are given in brackets after the names of the authors. The abbreviations are given in the following *table*:—

(A) = Amos	(N) = Neutra
(B) = Borrmann	(R) = Roussel
(D) = Deckart	(S) = Sauvé
(J, P & Q) = Jackson, Porter, and Quinby	(T) = Talke
(K) = Kölbing	(Z) = Zesas
(M & J) = Mauclairé and Jacoulet	

² The following 27 cases were *published* by Jackson, Porter and Quinby: the name of the observer of each case is given in brackets.

- Codman, J. G. Mumford, Withington, H. Jackson, Ames, J. C. Munro & E. H. Nichols, Thorndike); Jastrowitz (N), Jenni (Taravellier), Johst (M & J), De Jong 6, Jurgens (D).
- Kanis, Karcher, Kaufmann 2, Kendal-Franks, Kiliani, Kölbing 2, Körte (S), Köster (N), Köster (K) 2, Köster, Kussmaul (N).
- Labbé (J, P & Q), Lagane 9, Langerhans (T), Laveran (J, P & Q), Lecène (S), Leclerc & Beutter (M & J), Leclerc & Cotte, Leech, Lépine (J, P & Q), Lerat & Cléret, Lereboullet (T), Lewis & Rosenow, Lieblein (M & J) 2, Lilienthal (J, P & Q), Lindner (Z), Lobert (N), Lochte (N), London Hospital P. M.'s 30, Lorenz (J, P & Q), Lothrop (N) 3.
- McArthur, McCarthy, MacPherson, McWeeny, Mader (J, P & Q), Mauclaire & Jacoulet, Maygrier & Letulle, Maylard 2, Mengin (J, P & Q), Merkel (N), Merkel, Meyer (T), Meyer, Michaelis, Mignon & Dopfer, Mollard & Monod (M & J), Monnier & Suter (M & J) 2, Monro & Workman, Moos, Mouchet (Z), Moyes (N), Munro.
- Neumann (S), Niederstein, Nordenfelt (N), Nutt.
- Oppolzer (Z), Original 7, Osler 2, Osswald 3, Ott (T) 2.
- Packard (J, P & Q), Parensky (N) 3, Patal (S), Péron & Beaussenat (R), Picot, Picqué & Grégoire (T) 2, Pieper (N), Piery & Dumas (M & J), Pilliet (N), Pilliet (K), Pinner (N), Polya 4, Ponfick (D) 3, Putnam (N).
- Reitter (M & J), Reitzenstein, Richards, Riesman (J, P & Q), Riis (J, P & Q) 2, Rodger, Rolleston, Roper.
- Sachs (K), St Bartholomew's Hospital Museum Catalogue 2, Sauvé (Lenormant's case), Saxer (J, P & Q), Schlesinger (N), Schley, Schmorl (A), Schnitzler, Seitz (B), Senni (M & J), Shoemaker, Sievers (N), Sillitti (M & J) 2, Smith, W. G., Smith, F. J., Smith, G. M., Solieri (M & J), Späth (N), Spiegelberg, Sprengel 4, Stéphan (M & J), Straton, Stubbs (J, P & Q).
- Talke 3, Taravellier 2, Taylor, Tcherkavskaja, Thévenot & Rey 2, Thomas, Trübel (N) 4, Tschudy (M & J), Tschudy & Seitz (M & J), Tyson & Livingston.
- Ullmann (M & J).
- Van Hook (J, P & Q), Virchow 3.
- Wagner (T), Wakefield, Watson (N) 2, Welch & Flexner (J, P & Q), West, Westhoff (T), Whipple, Wikner (T), Wittmann (T), Woloschin, Wood, Woolsey (Kiliani).
- Zimmermann (T).

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